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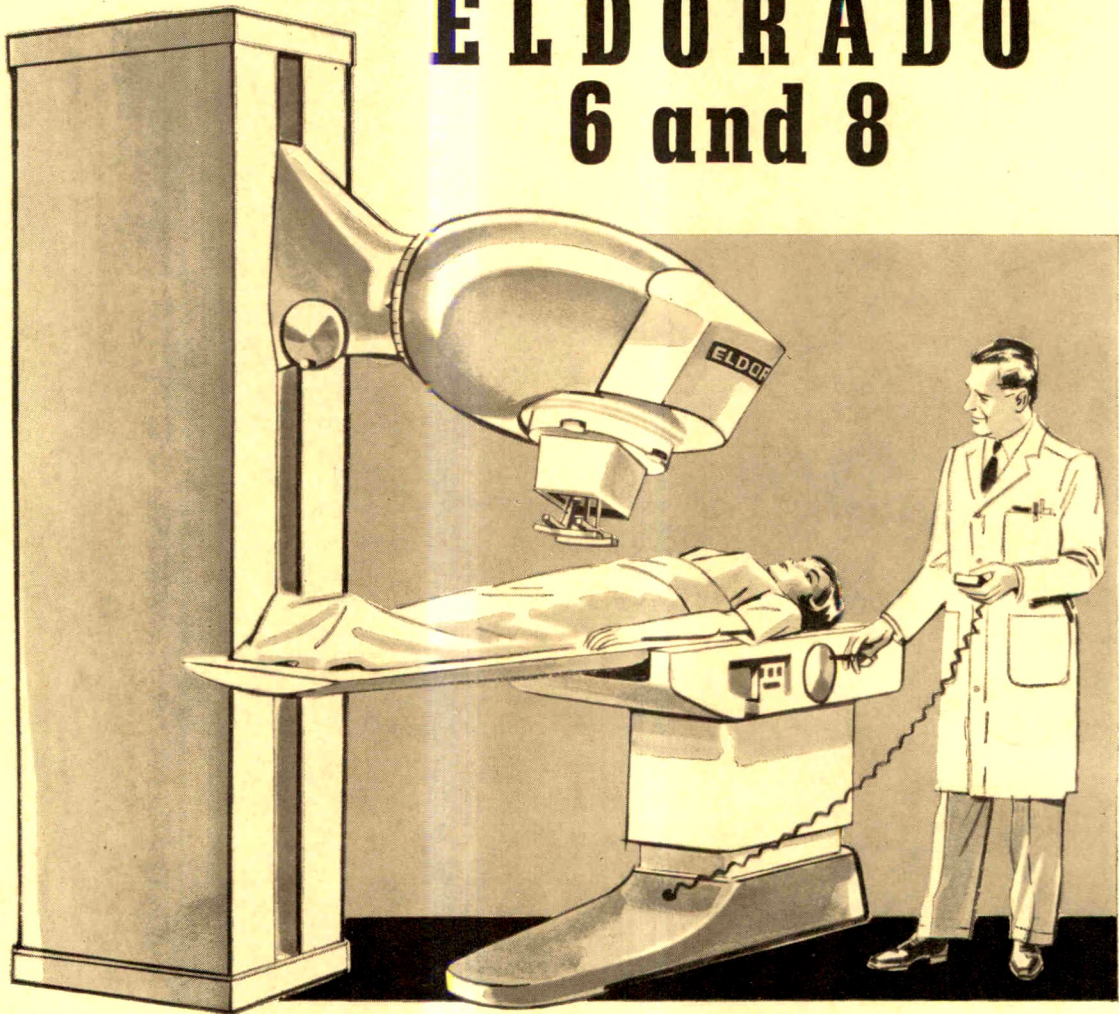
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Vol. 94

May, 1965

No. 1

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CONTENTS

<i>Percutaneous Catheterization via the Axillary Artery; A New Approach to Some Technical Roadblocks in Selective Arteriography.</i> PAUL ROY, M.D.	I
<i>Percutaneous Retrograde Brachial Arteriography; A Nonoperative, Non-catheter Technique.</i> ROBERT M. BAIRD, M.D. (LCDR, MC, USN), MARC S. LAPAYOWKER, M.D., FREDRICK MURTAGH, M.D., and MICHAEL SCOTT, M.D.	19
<i>Guided Angiography.</i> MANUEL VIAMONTE, JR., M.D., and ROBERT C. STEVENS.	30
<i>The Value of Catheter Arteriography in Evaluating Arterial Insufficiency.</i> J. L. CURRY, M.D., W. J. HOWLAND, M.D., and E. C. VOSS, JR., M.D.	40
<i>Angiocardiographic Diagnosis of Both Great Vessels Originating from Right Ventricle; Report of Thirteen Acyanotic Patients.</i> ISRAEL STEINBERG, M.D., and MARY ALLEN ENGLE, M.D.	45
<i>Anomalous Systemic Venous Connection to the Left Atrium or to a Pulmonary Vein.</i> HOOSHANG TAYBI, M.D., M.Sc. (PED.), GERALD J. KURLANDER, M.D., PAUL R. LURIE, M.D., and JOHN A. CAMPBELL, M.D.	62
<i>Infundibular Obstruction Secondary to Pulmonary Valvular Stenosis.</i> RICHARD G. LESTER, M.D., ROBERT T. OSTEEN, A.B., and ARVIN E. ROBINSON, M.D.	78
<i>Functional Pulmonary Atrisia; A Cause of Angiocardiographic Misinterpretation in Tetralogy of Fallot.</i> S. DAVID ROCKOFF, M.D., M.Sc. (RADIOLOGY), and JOSEPH GILBERT, M.D.	85
<i>Tetralogy of Fallot; A Detailed Angiocardiographic Study.</i> RICHARD G. LESTER, M.D., ARVIN E. ROBINSON, M.D., and ROBERT T. OSTEEN, A.B.	92
<i>Visualization of the Pulmonary Artery and Right Ventricle via a Patent Ductus Arteriosus; Report of 4 Illustrative Cases.</i> HARRY L. STEIN, M.D., and ISRAEL STEINBERG, M.D.	100

(Continued on page iv)

<i>Roentgenographic Manifestations of Endocardial Fibroelastosis.</i> I. S. JOHNSRUDE, M.D., AND LEWIS S. CAREY, M.D.	109
<i>Congenital Absence of the Left Pericardium.</i> BURTON S. TABAKIN, M.D., JOHN S. HANSON, M.D., JOHN P. TAMPAS, M.D., AND EDGAR J. CALDWELL, M.D.	122
<i>Thoracic Aortography; Intravenous and Selective Techniques.</i> ISRAEL STEINBERG, M.D., MORDECAI HALPERN, M.D., AND HARRY L. STEIN, M.D.	129
<i>Collateral Circulation in Aorto-Ilio-Femoral Occlusive Disease; As Demonstrated by a Unilateral Percutaneous Common Femoral Artery Needle Injection.</i> MARVIN J. FRIEDENBERG, M.D., AND CARLOS A. PEREZ, M.D.	145
<i>Collateral Vascular Pathways during Experimental Obstruction of Aorta and Inferior Vena Cava.</i> GUNTER GRUPP, M.D., INGRID L. GRUPP, M.D., AND HAROLD B. SPITZ, M.D.	159
<i>Intra-Osseous Costal Venography; Assessment of the Method in Studies on Portal Hypertension.</i> M. EIKEN, M.D., R. NIELSEN, M.D., AND H. BADEN, M.D.	172
<i>Arteriography of the Pancreas.</i> J. RÖSCH AND J. BRET	182
<i>Arteriography in Orthopedics.</i> MORDECAI HALPERN, M.D., AND ROBERT H. FREIBERGER, M.D.	194
<i>Ascending Phlebography in Fresh Thrombosis of the Lower Limb.</i> S. BORGSTRÖM, T. GREITZ, W. VAN DER LINDEN, J. MOLIN AND I. RUDICS.	207
<i>Selective Vasodilatation As an Aid to Angiography.</i> PAUL C. KAHN, M.D., AND ALLAN D. CALLOW, M.D.	213
<i>A Roentgenologic Approach to the Measurement of Venous Blood Flow.</i> C. GIANTURCO, M.D., AND F. R. STEGGERDA, PH.D.	221
<i>Segmental Occlusion of the Middle Cerebral Artery.</i> SANFORD J. LARSON, M.D., LEON LOVE, M.D., AND CHARLES TIMMONS, M.D.	223
<i>Vinylite Cast Preparations of Various Normal Organs.</i> B. MERINGOFF AND F. GARGANO, M.D.	230
<i>Darkroom Practice and Unnecessary Patient Exposure.</i> RUSSELL H. MORGAN AND HOWARD E. CHANEY	236
<i>Officers</i>	241
<i>Editorials</i>	
<i>The Cardiovascular Radiologist.</i> LEWIS S. CAREY, M.D.	242
<i>Henry Janney Walton, M.D., 1879-1965.</i> WALTER L. KILBY, M.D.	244
<i>Leo A. Nash, M.D., 1910-1964.</i> DAVID G. PUGH, M.D.	246
<i>News Items</i>	249
<i>Book Review</i>	250
<i>Books Received</i>	250
<i>Society Proceedings</i>	252
<i>Abstracts of Radiological Literature</i>	257

THE AMERICAN JOURNAL OF ROENTGENOLOGY, RADIUM THERAPY AND NUCLEAR MEDICINE

VOL. 94

JUNE, 1965

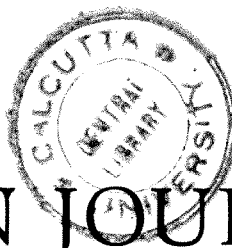
No. 2

CONTENTS

<i>Postirradiation Sialadenitis; A Study of the Clinical Features, Histopathologic Changes and Serum Enzyme Variations Following Irradiation of Human Salivary Glands.</i> HASKINS K. KASHIMA, M.D., WILLIAM R. KIRKHAM, PH.D., M.D., AND J. ROBERT ANDREWS, M.D.	271
<i>Juvenile Nasopharyngeal Fibroma; Roentgenologic Characteristics.</i> COLIN B. HOLMAN, M.D., AND W. EUGENE MILLER, M.D.	292
<i>The Evaluation of Pharyngeal Neuromuscular Disorders by Cinefluorography.</i> MARTIN W. DONNER, M.D., AND CHARLES I. SIEGEL, M.D.	299
<i>Allison and Johnstone's Anomaly.</i> JAMES THOMSON WRIGHT, M.D., D.M.R., M.C.R.A., M.R.C.P.E.	308
<i>Stricture of the Esophagus Due to Nasogastric Intubation.</i> IRVING WALDMAN, CAPTAIN, USAF MC, AND LEONARD BERLIN, CAPTAIN, USAF MC.	321
<i>Gastroesophageal Reflux Elicited While Drinking Water—(Water Siphonage Test); Its Clinical Correlation with Pyrosis.</i> JOSEPH F. LINSMAN, M.D.	325
<i>A Roentgenologic Study in Mediastinal Anatomy Afforded by Air in the Mid-Esophagus; A Normal Finding but a Potential Source of Diagnostic Error.</i> CHRISTIAN V. CIMMINO, M.D., F.F.R.	333
<i>Benign Tumor in Gastric Air Bubble.</i> SEYMOUR FISKE OCHSNER, M.D., AND GEORGE P. JANETOS, M.D.	337
<i>Partial Gastric Diverticula.</i> KAMILLO FLACHS, M.D., HENRY H. STELMAN, M.D., AND PAUL J. H. MATSUMOTO, M.D.	339
<i>The Roentgenographic Signs in Atrophic Gastritis and Gastric Atrophy.</i> JAMES F. MARTIN, M.D., THOMAS F. O'BRIEN, JR., M.D., IVAN L. HOLLEMAN, M.D., GEORGE H. WALL, M.D., AND J. L. DUQUE, M.D.	343
<i>Situs Inversus of All Organs except Stomach.</i> ARTHUR LIEBER, M.D., AND HAROLD D. ROSENBAUM, M.D.	353
<i>Roentgen Diagnosis of Duodenal Injuries.</i> MALCOLM C. HILL, M.B.	356
<i>Villous Adenoma of the Duodenum.</i> LEE A. MALMED, M.D., AND BERTRAM LEVIN, M.D.	362
<i>Syndrome of Jejunal Diverticulosis and Megaloblastic Anemia.</i> GERALD A. L. IRWIN, M.D.	366
<i>Small Bowel Obstruction Due to an Unusual Vermiform Meckel's Diverticulum Demonstrated by Barium Enema Study.</i> JULIUS H. GROLLMAN, M.D., AND DAVID SACHS, M.D.	370
<i>The Mass Sign in Primary Volvulus of the Small Intestine in Adults.</i> IRVING A. SHAUFFER, M.D., AND ERNEST J. FERRIS, M.D.	374

(Continued on page iv)

<i>Recognition of Ascariasis by Routine Chest or Abdomen Roentgenograms.</i>	
WILLIAM J. BEAN, M.D.	379
<i>Metastatic Carcinoma of the Small Bowel.</i>	
RICHARD H. MARSHAK, M.D., MANSHO T. KHLNANI, M.D., JOAN ELIASOPH, M.D., AND BERNARD S. WOLF, M.D.	385
<i>Hydrocele of the Spermatic Cord; With Roentgenographic Findings Simulating a Mucocele of the Appendix.</i>	
ERNEST J. FERRIS, M.D., AND IRVING A. SHAUFFER, M.D.	395
<i>Displacement of the Intestine by the Iliopsoas Muscle.</i>	
WILLIAM MARTEL, M.D.	399
<i>Roentgen Signs of Intestinal Necrosis.</i>	
LEO G. RIGLER, M.D., AND WILLIAM L. POGUE, M.D.	402
<i>The Fluid-Filled Colon in Acute Large Bowel Obstruction.</i>	
JOHN R. McIVER, M.B., CH.B., AND CONWAY DON, M.B., M.R.C.P., F.F.R.	410
<i>Abdominal Aortic and Iliac Graft Fistulae; Unusual Roentgenographic Find- ings.</i>	
ERNEST J. FERRIS, M.D., MARTA R. SZEGÖ KOLTAY, M.D., OSCAR P. KOLTAY, M.D., AND FARAG D. SCIAMMAS, M.D.	416
<i>Prostatic Carcinoma Involving the Rectum and Sigmoid Colon.</i>	
JOSHUA A. BECKER, M.D.	421
<i>Roentgenographic Findings in Zollinger-Ellison Syndrome.</i>	
MICHAEL M. MISSAKIAN, M.D., HARLEY C. CARLSON, M.D., AND KENNETH A. HUIZENGA, M.D.	429
<i>Roentgenologic-Pathologic Correlation of Resectable Carcinoma of the Pancreatico-Duodenal Region.</i>	
CARLOS A. PEREZ, M.D., WILLIAM E. POWERS, M.D., SUMNER HOLTZ, M.D., AND HARLAN J. SPJUT, M.D.	438
<i>Impression on the Duodenal Loop Resulting from Tumor of the Pancreas; A Roentgenographic Evaluation.</i>	
WARREN M. RUSSELL, M.D., AND ALEXANDER R. MARGULIS, M.D.	449
<i>Roentgenologic Possibilities in Spleen Diagnosis.</i>	
J. RÖSCH	453
<i>The Relative Accuracy of Estimation of Enlargement of the Liver and Spleen by Radiologic and Clinical Methods.</i>	
PAUL A. RIEMENSCHNEIDER, M.D., AND JOSEPH P. WHALEN, M.D.	462
<i>The Radioiodinated Rose Bengal Liver Scan As an Aid in the Differential Diagnosis of Jaundice.</i>	
W. R. EYLER, B. M. SCHUMAN, L. A. DU SAULT, AND R. E. HINSON	469
<i>A Case of Multi-Diverticular Cystic Dilatation of the Common and Hepatic Ducts.</i>	
GONZALO ESGUERRA-GÓMEZ, M.D., AND ENRIQUE RIVEROS- GAMBOA, M.D.	477
<i>The Roentgen Features of the Mirizzi Syndrome.</i>	
ARTHUR R. CLEMETT, M.D., AND ROBERT M. LOWMAN, M.D.	480
<i>Rapid Oral Cholecystography; Pharmacologic Assistance to the Use of Ora- grafin.</i>	
HARRY W. FISCHER, M.D., ALAN F. SCHROEDER, M.D., AND WILLIAM B. GALBRAITH, M.D.	484
<i>Comparative Evaluation of a New Oral Cholecystographic Agent U-12,031 with Telepaque.</i>	
PHILLIP H. MEYERS, M.D., CHARLES M. NICE, JR., M.D., PH.D., RAMON A. MOUTON, M.D., TIM CALDWELL, M.D., GEORGE R. MECKSTROTH, PH.D., S. THOMAS ELDER, PH.D., AND PHYLLIS J. MOSER, B.A., R.T.	491
<i>In Vitro and in Vivo Visualization of Biliary Calculi.</i>	
TOM W. STAPLE, M.D. AND WILLIAM H. McALISTER, M.D.	495
<i>Officers</i>	500
<i>Editorial</i>	
<i>The Forty-seventh Annual Meeting of the American Radium Society.</i>	
HARRY HAUSER, M.D.	501
<i>News Items</i>	506
<i>Book Reviews</i>	507
<i>Books Received</i>	508
<i>Abstracts of Radiological Literature</i>	510



THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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No. 1

PERCUTANEOUS CATHETERIZATION VIA THE AXILLARY ARTERY*

A NEW APPROACH TO SOME TECHNICAL ROADBLOCKS IN SELECTIVE ARTERIOGRAPHY

By PAUL ROY, M.D.†

MONTREAL, QUEBEC, CANADA

WITHIN the past few years great strides have been made in cardiac and vascular radiology, but unlimited possibilities for further development remain. Since ease of performance and the low morbidity of a method are equally as important as the quality and quantity of information gained, it must be borne in mind that the technical procedures in this field are still in the creative and adjustment stages; newer approaches must be developed to overcome problems that have arisen during the use of existing techniques.

At the present time, Seldinger's method^{2,3} of percutaneous catheterization of the femoral artery by means of a radiopaque tubing^{11,12} has largely replaced direct aortography and intravenous opacification of the arterial system.^{26,27} The dangers of direct needle injection are avoided and selective opacification of the aorta at the desired level may be realized with precision under fluoroscopic control. Furthermore,

catheterization via this route has permitted selective arteriography¹⁰ of the renal, mesenteric, celiac, coronary, subclavian, carotid and vertebral arteries. Unfortunately, however, certain pathologic conditions prohibit utilization of the femoral vessels.^{2,3} In the presence of a Leriche syndrome, tortuosity or stenosis of the femoral or iliac arteries, and aneurysm of the abdominal aorta, retrograde femoral catheterization becomes impractical or dangerous. In addition, due to their parallel course to the aorta, the celiac and superior mesenteric arteries may defy selective catheterization by the retrograde femoral route, even when the catheter is of small caliber and has a hook-shaped end (Fig. 1, *A*, *B* and *C*). For the same reason, selective unilateral renal arteriography is sometimes difficult or impossible to achieve.

Problems may also be encountered at the thoracic level when the catheter is introduced through the femoral artery. Access

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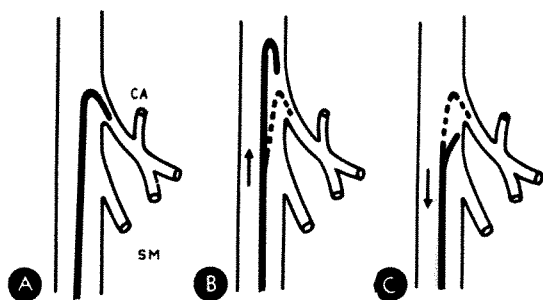


FIG. 1. (A, B and C) Pitfalls in selective celiac or superior mesenteric catheterization by the femoral route. Nearly vertical direction of vessels requires such acute angulation of catheter that it cannot penetrate a sufficient distance. Additional maneuvers make it slide back into the aorta. CA—celiac artery; SM—superior mesenteric artery.

to the subclavian or common carotid artery on the left and the innominate artery on the right is often impossible, particularly in older people where the arteries at the base of the neck become elongated and tortuous due to arteriosclerosis (Fig. 2, *A* and *B*; and 3, *A*, *B* and *C*). The catheter, which has followed the aortic lumen through nearly all of its length, does not respond satisfactorily to the operator's maneuvers and frequently cannot be guided into the abnormal angle formed by the arteriosclerotic vessels and the arch of the aorta. This is particularly true when the aorta is markedly uncoiled (Fig. 4, *A* and *B*). Certain alterations in the curvature of the catheter will sometimes permit these obstacles to be overcome, but, in a great many instances the femoral approach results in failure.

In an attempt to circumvent these difficulties, some workers^{2,15,16,17,18} have resorted to catheterization of the radial or brachial arteries (Fig. 5). However, in using this approach, even greater problems are commonly encountered. Since these vessels are smaller in caliber than the femoral artery, they are less tolerant to trauma^{2,5,28} and sometimes react with such intense spasm that even needle puncture becomes difficult. When introduction of the catheter is possible, spasm also fre-

quently immobilizes the tube so that the guiding movements of the operator are ineffective. Successful catheterization may be followed by marked spastic phenomena which make withdrawal of the catheter laborious and may result in such serious complications as thrombosis.

For these reasons percutaneous puncture of the axillary artery was begun in the Department of Radiology of the Hôtel-Dieu Hospital in January, 1961. In spite of a few and discouraging attempts by Peirce¹⁴ and by Seldinger,²³ it was assumed that the larger and readily palpable vessel would be more tolerant to trauma. In a series of 3,476 vascular studies performed during a 3 year period, the author has employed the axillary artery in 475 different cases for selective roentgenologic exploration of the aorta and its subdivisions. Other workers have become aware of our procedure through personal communications, preliminary reports^{19,20} and lectures at various radiologic meetings.²¹ Some have adopted the procedure and their experience has paralleled ours; the axillary artery represents a convenient and safe

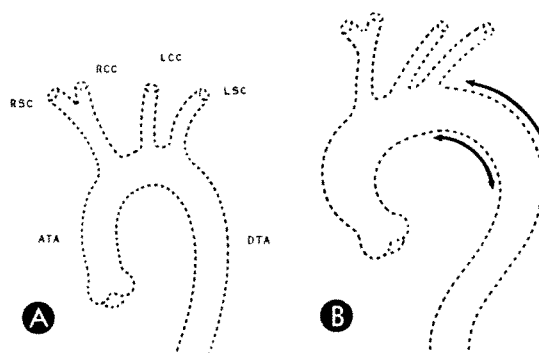


FIG. 2. (A) Relative position of the arteries of the base of the neck, originating from the normal aortic arch (left anterior oblique view). ATA—ascending thoracic aorta; RSC—right subclavian artery; RCC—right common carotid artery; LCC—left common carotid artery; LSC—left subclavian artery; DTA—descending thoracic aorta. (B) In arteriosclerosis, elongation and uncoiling of the aortic arch are most often marked in its distal portion. Consequently, the vessels of the base of the neck are displaced by their proximal segment towards the anterior mediastinum.

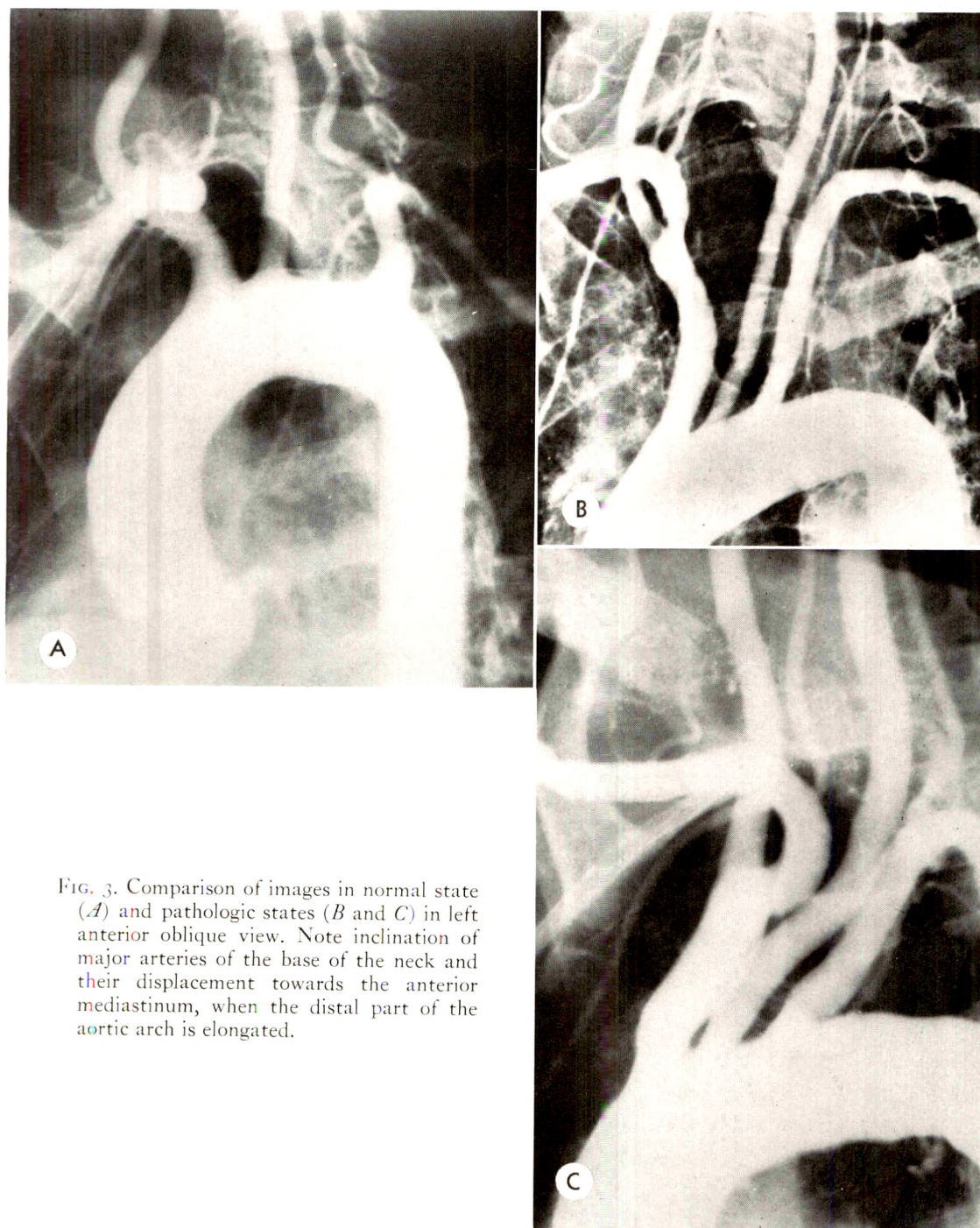


FIG. 3. Comparison of images in normal state (A) and pathologic states (B and C) in left anterior oblique view. Note inclination of major arteries of the base of the neck and their displacement towards the anterior mediastinum, when the distal part of the aortic arch is elongated.

approach to the aorta and its subdivisions.^{1,3,9,13} It provides a satisfactory alternative to the femoral route in the presence of certain pathologic states, and is probably preferable for opacification of the vessels of the upper extremities or head and neck.

PUNCTURE OF THE AXILLARY ARTERY: TECHNIQUE

With the arm extended in supination and abducted nearly 135° from the body, the axillary artery can be palpated immedi-

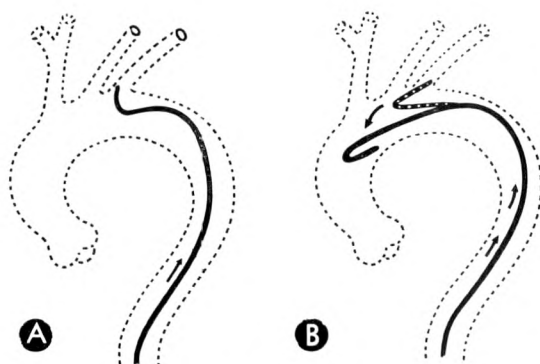


FIG. 4. Attempt at selective catheterization of the left subclavian artery by the femoral approach. The aortic arch is elongated and uncoiled by arteriosclerosis. (A) Since the direction of the vessels is not favorable, the tip of the catheter cannot penetrate the left subclavian artery, either because it comes in immediate contact with the wall of this vessel or it forms a loop in the aortic arch. (B) By attempting further penetration into the selected vessel, the tip of the tubing invariably slides back into the ascending aorta. Alternately, if engagement is not complete, back push on the catheter during rapid injection of opaque material results in the same outcome.

ately posterior to the pectoral muscles (Fig. 6). The skin and tissues adjacent to the artery are infiltrated with a local anesthetic such as 2 per cent xylocaine. At this preliminary stage, the needle tip may touch a few branches of the brachial plexus

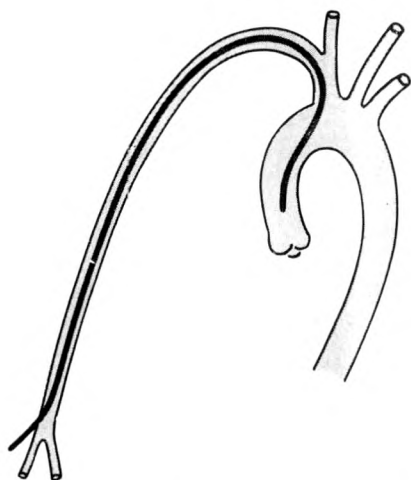


FIG. 5. Catheterization of the thoracic aorta by the brachial approach.

(Fig. 7). The patient, previously warned of this possibility, immediately indicates the pain felt, and the operator utilizes the opportunity to perform a partial nerve block. A characteristic numbness from arm to hand in the ulnar, median, or radial distributions is experienced usually with varying degrees of motor weakness.

A tiny superficial incision is then made in the skin with the tip of a scalpel. The tissues are delicately dissected with the blunt end of a forceps, producing a small wound 2 or 3 mm. in diameter through which the artery may be reached without interference from the overlying soft tissues. An Ödman needle of small caliber with the internal probe withdrawn is directed medially and upward in the line of the artery at an angle of 45° from the skin surface (Fig. 8A). Penetration of the soft tissue must be performed slowly and gently with alternating clockwise and counter-clockwise rotations until the arterial pulse is transmitted to the fingers of the operator. The needle is then pushed forward until the arterial wall is perforated. With proper insertion, a few drops of blood are immediately seen at the orifice of the inner cannula. The latter is then withdrawn and a jet of blood is seen with each systolic phase. The remaining outer trocar is advanced into the lumen of the artery at an angle of approximately 10° ; *i.e.*, the plane of the



FIG. 6. Needle penetrates the axilla.

trocár roughly parallels the surface of the skin (Fig. 8B). To avoid blockage, the cannula should be introduced in such a fashion that the free flow of blood is not impeded. For upper limb arteriography, the needle is connected by means of a large polyethylene tubing to a syringe containing the contrast medium. The connection tubing should have some degree of flexibility, but be sufficiently resistant to withstand the injection pressure. Coupling is carried out by means of Luer-Lok adaptors. When it is necessary to delay the injection or await the processing of films, patency of the cannula is maintained by a slow drip of an isotonic sodium chloride solution under pressure.

For percutaneous catheterization (Fig. 9), arterial puncture is performed in an identical manner, following which the trocar is replaced by tubing as described by Seldinger.

Following completion of the procedure and withdrawal of the catheter, the operator or his assistant exerts firm manual pressure on the axilla for 10 to 15 minutes. This is essential to prevent formation of a hematoma.

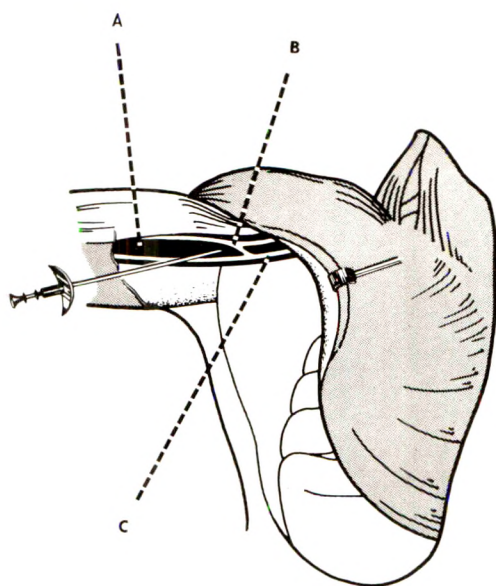


FIG. 7. Site of percutaneous puncture for axillary approach. A—axillary artery; B and C—components of the brachial plexus.

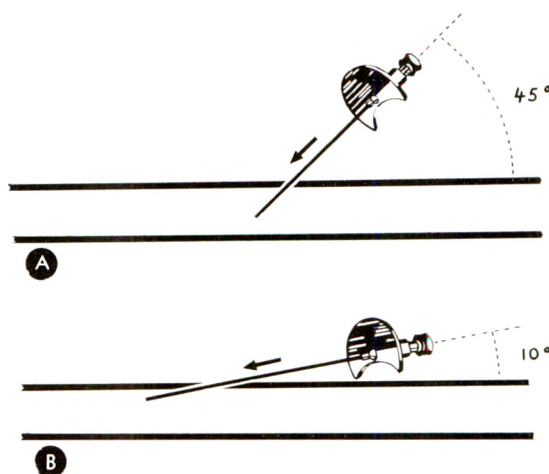


FIG. 8. (A) The needle is first introduced in the artery, forming with it an angle of 45° . (B) The needle is then lowered to an angle of approximately 10° , to ensure further penetration in the axis of the arterial lumen.

APPLICATIONS OF AXILLARY ARTERY PUNCTURE A. UPPER EXTREMITIES

Our first attempts were limited to arteriography of the upper limb by percutaneous puncture of the axillary artery and direct needle injection of 30 cc. of 60 per cent renografin.^{19,20} The injection was carried out within 2 seconds under an average pressure of 25 pounds per square inch. With the needle properly positioned in the axis of the lumen of the vessel, it was noted that retrograde reflux of the opaque substance would reach the innominate artery on the right or the proximal portion of the



FIG. 9. Catheter in the axillary artery.

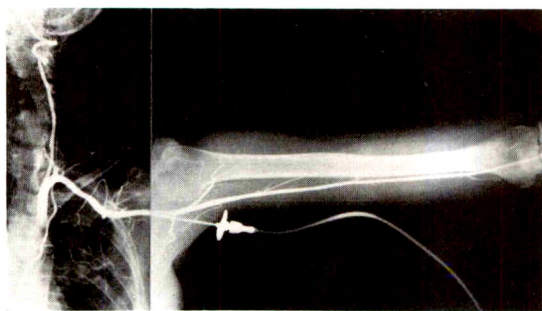


FIG. 10. Direct arteriography of the left upper limb by percutaneous puncture of the axillary artery demonstrating a considerable back flow of opaque material into the subclavian artery and its collaterals, particularly the vertebral artery.

subclavian artery on the left (Fig. 10). Consequently, with this technique, it was possible during arteriography of the left upper limb to commonly visualize the vertebral artery and the collaterals of the subclavian artery. On the right, the right common, internal and external carotid arteries as well as the right vertebral arteries are usually visualized on the film exposed immediately at the end of the injection. In the few seconds following this, excellent opacification of the arteries of the arm, forearm, and finally the hand and fingers (Fig. 11) can be obtained. Obstructions of any type with attendant collateral circulation are readily demonstrable.

Since this technique proved so successful, we did not hesitate to employ the axillary artery for percutaneous arterial catheterization (Table I). During 3 years starting in January, 1961, this approach



FIG. 11. Arteriography of the right hand. Direct injection of the opaque material by needle in the axillary artery. Raynaud's disease affecting the fourth finger and showing segmental occlusion in the proper digital arteries. Note the considerable delay in opacification of the vascular network at the tip of the same finger.

has been employed for 475 arteriographic examinations. This experience has permitted us to explore and record all potentialities of the procedure and to selectively

TABLE I
AXILLARY APPROACH TO ARTERIOGRAPHY
(Cases examined in 3 years)

Applications	Right Approach	Left Approach	Total
Direct Axillary Arteriography (with a needle)	40	39	79
Selective Aortography or Arteriography (with a catheter)	170	226	396
Total	210	265	475

TABLE II
RANGE OF APPLICATIONS OF PERCUTANEOUS AXILLARY ARTERY CATHETERIZATION

Approach	Target for Opaque Injection	Type of Examination
Right Axillary Artery	Left ventricle	Levoangiocardiology
	Ascending aorta (4 or 5 cm. above the aortic valve)	Thoracic aortography Simultaneous, bilateral carotid and vertebral arteriography Morphologic and functional study of the aortic valve
	Ascending aorta (Valsalva sinuses)	Bilateral coronary arteriography Selective coronary arteriography
	Innominate artery Right subclavian artery	Right carotid and vertebral arteriography Basilar and right carotid arteriography
Left Axillary Artery	Left ventricle	Levoangiocardiology
	Ascending aorta (4 or 5 cm. above the aortic valve)	Thoracic aortography Simultaneous, bilateral carotid and vertebral arteriography Morphologic and functional study of the aortic valve
	Ascending aorta (Valsalva sinuses)	Bilateral coronary arteriography Selective coronary arteriography
	Left subclavian artery	Left vertebral arteriography Basilar arteriography
	Proximal abdominal aorta	Abdominal aortography
	Celiac artery	Selective celiac arteriography Selective splenic arteriography Selective hepatic arteriography
	Renal artery (right or left)	Selective renal arteriography
	Mesenteric artery (superior or inferior)	Selective mesenteric arteriography
	Distal abdominal aorta	Bilateral aorto-iliac arteriography Bilateral lower limb aorto-arteriography
	Iliac artery	Selective iliac arteriography (common, external, internal)
	Femoral artery	Selective femoral arteriography

choose the right or left axillary artery depending upon the information desired (Table II).

B. VESSELS OF THE HEAD AND NECK

Although direct puncture of the com-

mon or internal carotid arteries remains the most frequently utilized and satisfactory method of cerebral angiography, it is inadequate for visualizing the origin of the common carotid arteries and is useful in visualizing the posterior cerebral arteries in

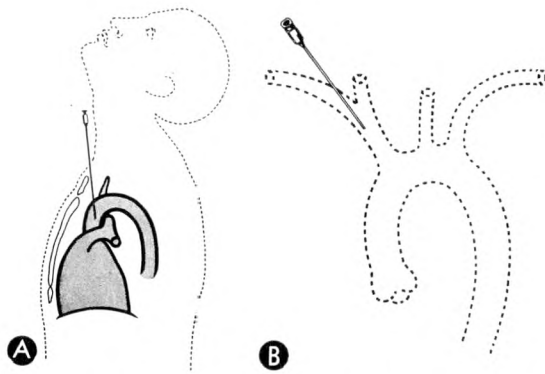


FIG. 12. (A) Suprasternal puncture of the aortic arch for thoracic aortography. (B) Percutaneous puncture of the subclavian artery for direct arteriography.

only 30 per cent of cases. Total opacifications of the carotid and vertebral systems may be attained by suprasternal aortography,⁴ but this direct method (Fig. 12A) may require general anesthesia and carries the risk of an uncontrolled hematoma or intramural injection with dissection of the arterial wall. Because of the risk of dislodging the needle or tearing the vessel, alteration of the position of the patient during examination is also difficult and documentation may be inadequate. Direct injection into the subclavian artery (Fig. 12B) has been used as an alternative method and for the most part results in

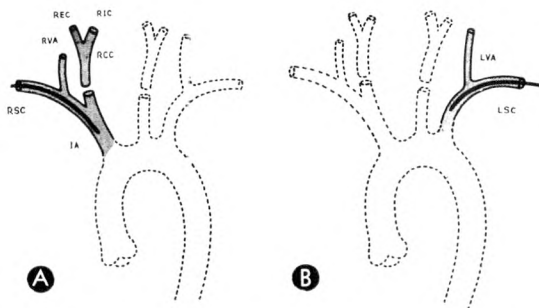


FIG. 13. (A) Catheter in the innominate artery. RSC—right subclavian artery; RVA—right vertebral artery; IA—innominate artery; RCC—right common carotid artery; REC—right external carotid artery; RIC—right internal carotid artery. (B) Catheter in the left subclavian artery. LSC—left subclavian artery; LVA—left vertebral artery.

satisfactory arteriograms.⁸ However, technical difficulties may sometimes be the cause of a perforation of the apical pleura with the subsequent development of a hemo- or pneumothorax and extravasation of the opaque medium may result in damage to the brachial plexus. Percutaneous puncture of the vertebral artery with direct injection of the opaque medium is, therefore, the most commonly employed approach for visualization of the posterior cerebral circulation.^{6,7,24,25} Unfortunately, this is the most dangerous as well as the most direct method. Injury or prolonged spasm of the vertebral artery may endanger the life of the patient due to sudden ischemia of the mid-brain (mesencephalon), pons or medulla oblongata, particularly



FIG. 14. Demonstration of the cervical segment of the left vertebral artery by injection of contrast material in the subclavian artery, after percutaneous catheterization via the left axillary artery. Slight narrowing at the origin of the vertebral artery.

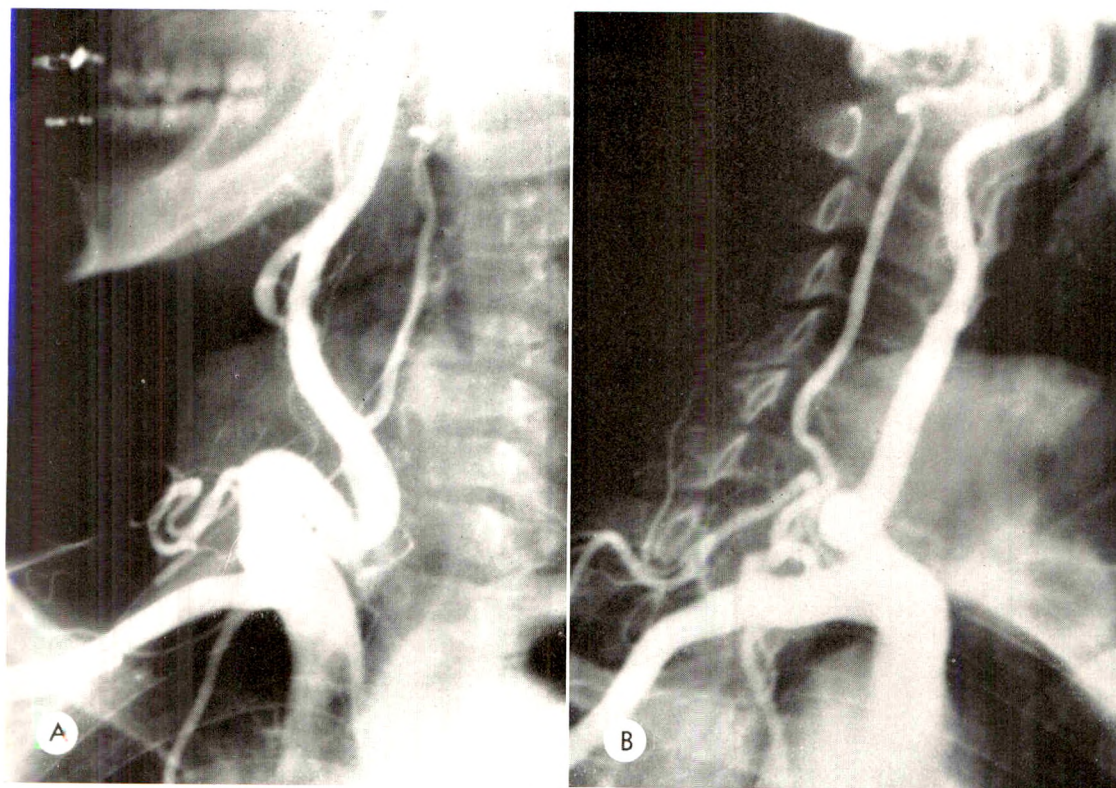


FIG. 15. Demonstration of vascular structures to the right, at the level of the neck, by injection of contrast agent in the innominate artery via right axillary percutaneous catheterization. Considerable arteriosclerotic elongation of the right common carotid artery which forms a double loop. During the clinical investigation, this was felt as a pulsatile mass and was misinterpreted as an aneurysm. (A) Left anterior oblique view. (B) Right anterior oblique view.

when agenesis or obliteration of the contralateral vertebral artery is present. Fatal outcomes have also been reported in the presence of deficient circulation in the posterior portion of the circle of Willis.

The potential hazards of the brachial and radial routes have previously been described and the advantages of the axillary approach noted.

Vertebral and basilar arteriography is performed by rapid injection of the contrast medium (20 cc. of 60 per cent or 76 per cent renografin in 2 or 3 seconds) while the tip of the catheter is placed in the proximal portion of the subclavian artery (Fig. 13B; and 14). On the right, injection in the innominate artery yields simultaneous carotid arteriography and vertebral-basilar opacification (Fig. 13A; 15, A and B; and 16). As emphasized for arteriography of up-

per limbs, direct and rapid needle injection of opaque material against the blood flow in the right axillary artery usually gives an excellent visualization of the right carotid system as well as of the vertebral and basilar circulation in the head and neck. Injection made in the left axillary artery also results in a clear demonstration of the left vertebral artery in the cervical region together with the basilar artery in the cranium.

For selective vertebral artery catheterization²¹ and basilar arteriography (Fig. 17, A and B; and 18, A and B), we have on several occasions successfully employed a fine, opaque polyethylene tubing with a slightly curved extremity. From the axillary artery, the proximal segment of the vertebral artery was easily reached on 31 occasions in a series of 41 attempts. How-

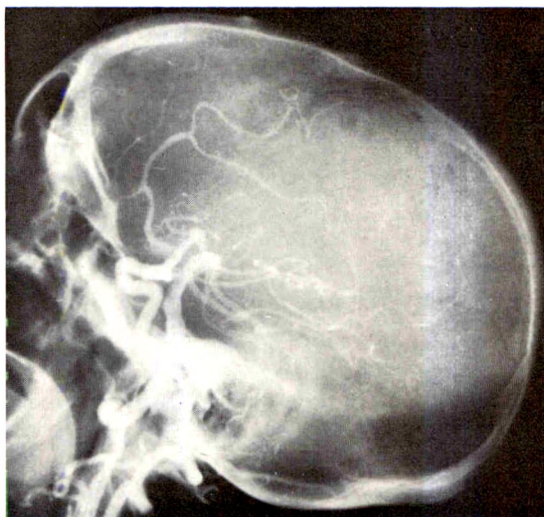


FIG. 16. Lateral view of the skull. Simultaneous basilar and right carotid opacification. Contrast material was injected in the innominate artery via right axillary percutaneous catheterization. Gross enlargement of the basilar and the right middle cerebral arteries due to arteriosclerotic deterioration of their walls. Some dilution of the opaque material in the basilar artery is probably caused by unopacified blood coming from the left vertebral artery.

ever, when the procedure results in a failure on one side, it will usually succeed on the other side.

Simultaneous visualization of the main arteries of the neck, that is, the innominate artery, subclavian arteries, carotid and vertebral arteries, from their origin to the base of the skull, is obtained routinely by rapid and massive injection of 50 cc. of 85 per cent cardiografin or 80 per cent angioconray in 2 seconds. The tip of the catheter in such cases lies in the ascending aorta midway between the aortic valve and the innominate artery (Fig. 19, *A* and *B*; and 20).

C. THORACIC AORTA

Since the innominate artery is nearly vertical to the horizontal portion of the aortic arch, it is rarely possible to catheterize the descending aorta via the right axillary approach; catheterization of the ascending aorta, on the other hand, is easily done (Fig. 19*A*). By employing the left axillary approach, the catheter readily

enters the ascending aorta (Fig. 19*B*) or the descending aorta (Fig. 21) as desired, provided the tip of the catheter is slightly curved so that it may be maneuvered by rotation and directed to one side or the other under fluoroscopic control.

Our attempts at selective catheterization of the coronary arteries by either axillary route have been few and sometimes fruitless. It is hoped that with further experience and certain modifications in the curvature and shape of the tip of the catheter, such examinations will be more successful in the future.

D. ABDOMINAL AORTA

In our opinion, the translumbar approach for abdominal aortography should be reserved only for those cases where a Leriche syndrome is suspected, provided the aorta is punctured well below the renal arteries. Under such circumstances, the only reasons in favor of the translumbar method are the economy of time and ease of execution. Nevertheless, it seems more logical and less hazardous to inject the opaque material in the abdominal aorta after catheterization by the percutaneous axillary approach (Fig. 21; and 22).

The most spectacular results have been obtained from selective arteriography of the celiac artery (13 cases). In fact, by its vertical direction, often nearly parallel to the aorta, this vessel lends itself admirably to the introduction of a catheter directed

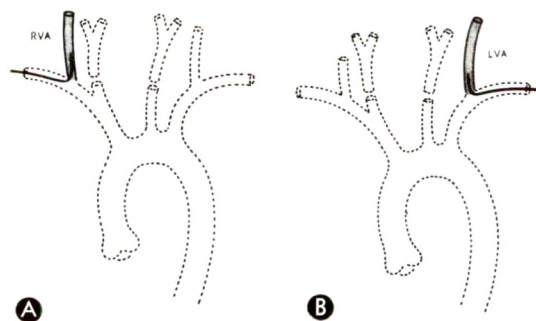


FIG. 17. Selective catheterization of the vertebral arteries. (*A*) Right axillary approach. (*B*) Left axillary approach. RVA—right vertebral artery; LVA—left vertebral artery.

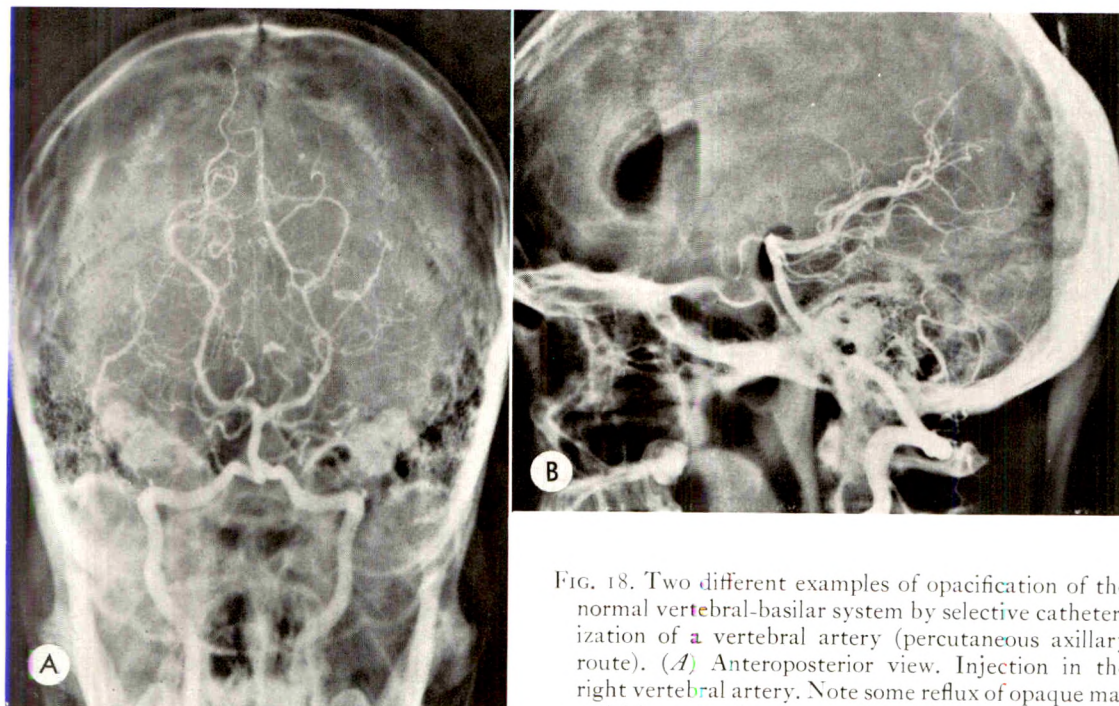


FIG. 18. Two different examples of opacification of the normal vertebral-basilar system by selective catheterization of a vertebral artery (percutaneous axillary route). (A) Anteroposterior view. Injection in the right vertebral artery. Note some reflux of opaque material into the distal part of the left vertebral artery. (B) Lateral view. In this case, injection was made in the left vertebral artery, 24 hours after pneumoencephalography.

caudally along the descending aorta (Fig. 23, A and B; and 24, A, B and C). In most instances, by further introduction of the catheter, the tip spontaneously enters the splenic artery; with a few rotating movements it may be made to enter the hepatic artery.

The same anatomic factors explain the ease with which selective mesenteric arteriography (Fig. 25A) may be performed (8 cases). Selective renal catheterization (Fig. 25B) is achieved with equal ease using the left axillary route (18 cases).

TECHNICAL PROBLEMS AND RESULTS

A retrospective study of a series of 475 examinations indicates the number of regions accessible by such a procedure. Comparison of successful examinations and failures permits evaluation which allows an anticipation of the technical difficulties which may be encountered, particularly at certain sites where arterial pathology was an obstacle to the passage of the catheter (Table III). A few of the failures via the axillary ap-

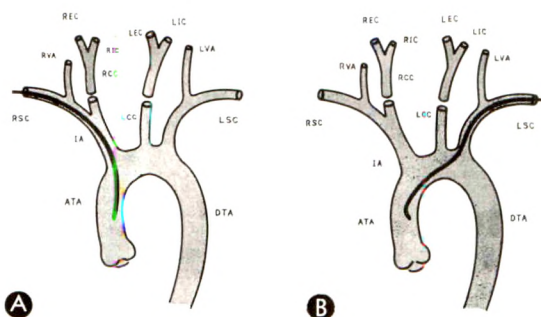


FIG. 19. Catheterization of the ascending thoracic aorta by the axillary approach. ATA—ascending thoracic aorta; IA—innominate artery; RSC—right subclavian artery; RVA—right vertebral artery; RCC—right common carotid artery; REC—right external carotid artery; RIC—right internal carotid artery; LCC—left common carotid artery; LEC—left external carotid artery; LIC—left internal carotid artery; LVA—left vertebral artery; LSC—left subclavian artery; DTA—descending thoracic aorta. (A) The right axillary route facilitates the procedure because of the favorable direction of the innominate artery, the emergence axis of which is almost in the prolongation of the proximal aorta; the catheter has no sharp curves to pass. (B) When stenosis or tortuosity of the innominate artery prevents catheterization of the ascending aorta, this may be done by the left axillary route.

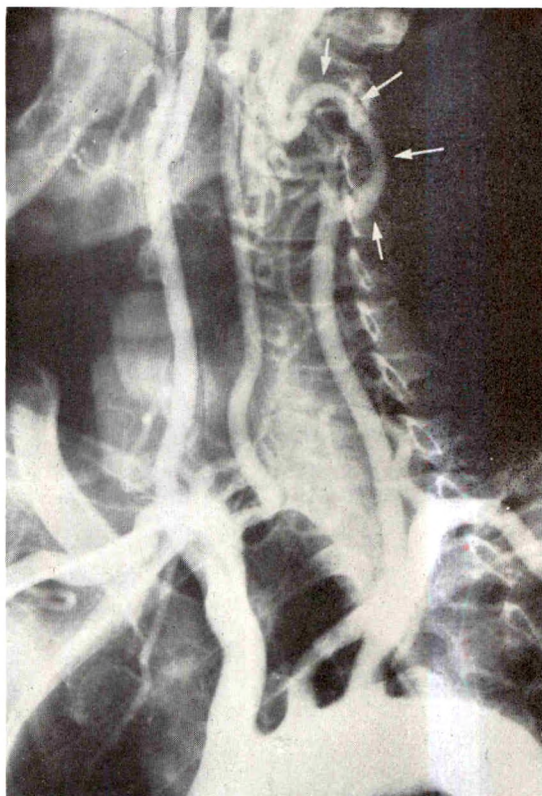


FIG. 20. Thoracic aortography (left anterior oblique position) after rapid injection of 50 cc. of 85 per cent cardiografin in the ascending aorta by catheterization via right percutaneous axillary approach. From reader's left to right, originating from the aortic arch: innominate, left common carotid and left subclavian arteries. Notice the loop formed by elongation of the left internal carotid artery (arrows). Clinically, this corresponded to a pulsatile mass mistaken for a cervical aneurysm. The right vertebral artery has a large caliber, while the left one is obliterated or absent.

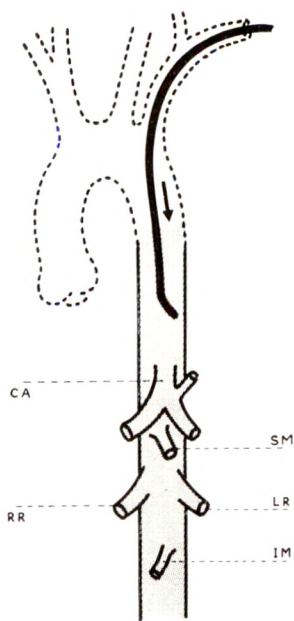


FIG. 21. Catheterization of the descending aorta for abdominal aortography and selective arteriography of abdominal viscera. Introduction by the

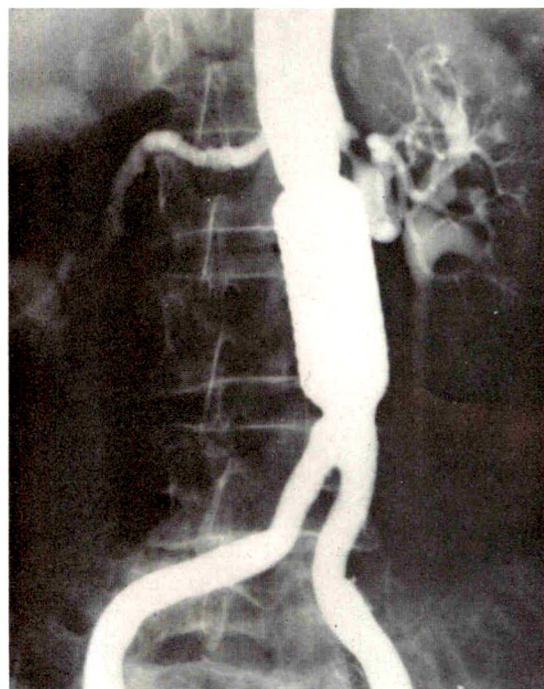


FIG. 22. Synthetic graft replacing a segment of the abdominal aorta. Injection of opaque material was performed after abdominal aorta catheterization by the left percutaneous axillary route. Marked stenosis of the left renal artery. Modifications of the right renal artery (fibromuscular rings?).

left axillary route. CA—celiac artery; SM—superior mesenteric artery; RR—right renal artery; LR—left renal artery; IM—inferior mesenteric artery.

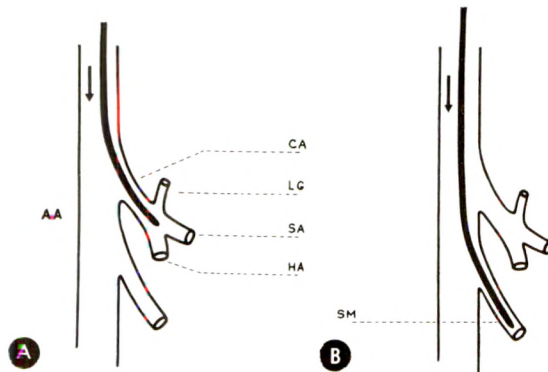


FIG. 23. (A and B) Because of their direction which is often almost vertical and nearly parallel to the aorta, the celiac and mesenteric arteries are easily catheterized by the descending route, rather than by the ascending one (Fig. 1, A, B and C). The axillary approach avoids the impediments of the femoral route. AA—abdominal aorta; CA—celiac artery; LG—left gastric artery; SA—splenic artery; HA—hepatic artery; SM—superior mesenteric artery.

proach were associated with previous unsuccessful attempts at catheterization of the femoral artery. It is to be emphasized that in only 3 cases (0.6 per cent) was axillary puncture abandoned because of insurmountable difficulties, although, at the onset, maneuvers were more laborious due to our inexperience in introducing a needle into this segment of the arterial system. One must admit that the axillary puncture, in comparison with the femoral, sometimes requires more patience and greater skill.

Stenosis of the axillary or subclavian arteries manifestly constitutes an obstacle to the axillary approach. In certain cases of advanced arteriosclerosis, the right innominate artery may be elongated or looped to the point of impeding the passage of the catheter into the aorta. In other cases, subclavian or carotid and vertebral arteriography

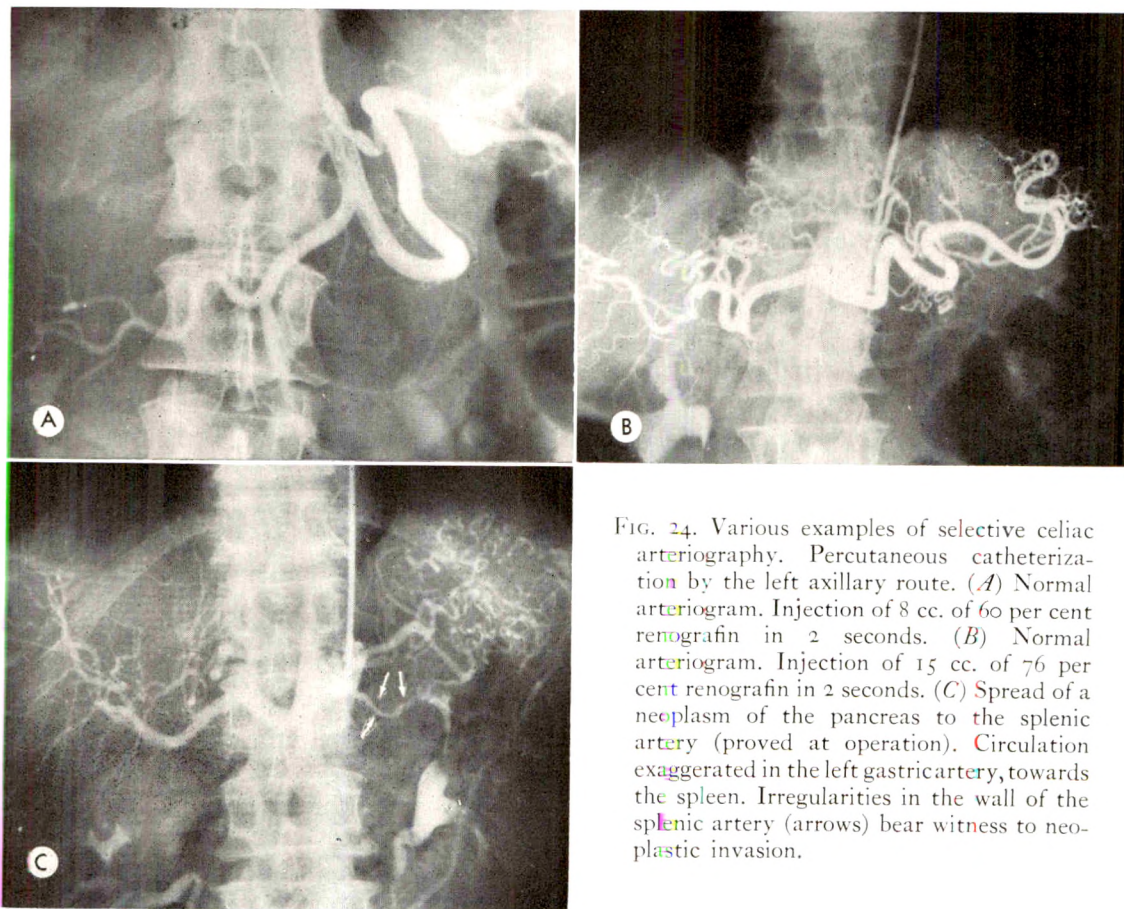


FIG. 24. Various examples of selective celiac arteriography. Percutaneous catheterization by the left axillary route. (A) Normal arteriogram. Injection of 8 cc. of 60 per cent renografin in 2 seconds. (B) Normal arteriogram. Injection of 15 cc. of 76 per cent renografin in 2 seconds. (C) Spread of a neoplasm of the pancreas to the splenic artery (proved at operation). Circulation exaggerated in the left gastric artery, towards the spleen. Irregularities in the wall of the splenic artery (arrows) bear witness to neoplastic invasion.

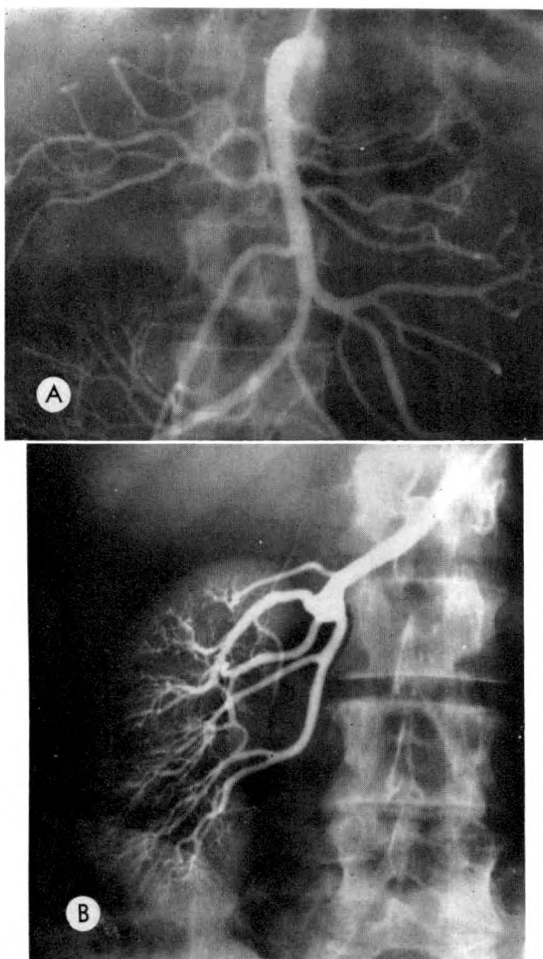


FIG. 25. Selective catheterization of abdominal visceral arteries by percutaneous left axillary approach. (A) Normal selective superior mesenteric arteriogram. (B) Right selective renal arteriogram.

raphy can be obtained only by rapid direct injection into the right axillary artery. Reflux of the opaque material usually suffices to give satisfactory images.

In 23 cases (5.8 per cent), manipulation of the catheter was definitely hindered by tortuosities, sharp angulation, or narrowing of the innominate artery or right or left subclavian arteries. In 10 instances (2.5 per cent), minor parietal damage resulted from attempts to bypass such areas as indicated by partial extravasation of the opaque medium during the test injection. Follow-up of these patients after such failures re-

vealed no clinical sequelae from the minor trauma.

If the catheter has to be advanced into the ascending aorta or the left chambers of the heart and if obstacles prevent the right axillary approach, the left axillary route may be used as a satisfactory alternative approach.

When the aortic arch is excessively uncoiled by arteriosclerotic elongation, the left subclavian artery often originates more anteriorly and to the right, so that the catheter spontaneously enters the ascending aorta (Fig. 26, A and B). For this reason, if it is necessary to reach the descending aorta, one must use a catheter with a markedly curved tip. On occasion, it may be necessary to facilitate this maneuver by passing a guide wire with a properly curved flexible tip. Fluoroscopic control is more effective in a left anterior oblique view by having the patient tilted to the right, or preferably, if equipment permits (Fig. 27), by directing the roentgen-ray beam along an oblique plane while the patient remains in a comfortable supine position. In spite of such circumspection and technical refinements, there remains a small number of cases where introduction of a catheter into the thoracic portion of the descending aorta or the abdominal aorta via the left axillary approach is impossible.

Fourteen different attempts at catheterization of the abdominal aorta via the left axillary approach have failed because of excessive elongation of the aortic arch. On each occasion, in spite of numerous modifications made in the curvature of the catheters and guide wire, the tubing invariably entered the ascending aorta (Fig. 26B). It should be noted, however, that we have succeeded in 154 similar cases when the alterations of the thoracic aorta were probably less marked. Failures were also less numerous after we gained more experience.

Because of its peculiar anatomic site, the left common carotid artery is seldom reached by catheterization through the femoral approach. The vessel is still less ac-

TABLE III

COMPARISON BETWEEN SUCCESS AND FAILURE OF PERCUTANEOUS AXILLARY ARTERY CATHETERIZATION*
(3 year experience)

Axillary Approach	Destination of Catheter Tip	No. of Cases	Success	Failure (total: 46)				
				Axillary Puncture	Obstacles on the Arterial Pathway		Innominate Artery	Aortic Arch
					Right	Left		
Right (170 cases)	Right subclavian or innominate artery	58	54	1	2		1	
	Ascending aorta	105	91	1	7		6	0
	Left heart							
	Descending thoracic aorta Abdominal aorta Abdominal visceral artery	7	4	0	0		0	3
Left (226 cases)	Left subclavian artery	25	23	1		1		
	Ascending aorta	30	24	0		3		3
	Left heart							
	Descending thoracic aorta Abdominal aorta Abdominal visceral artery	171	154	0		3		14
Total		396	350	3	9	7	7	20

* These data do not include 79 cases of upper limb arteriography performed by direct opaque injection with a needle in the axillary artery.

cessible by the left or right axillary routes. Visualization of the left common carotid in its cervical portion is, therefore, usually possible only by injection of opaque material into the ascending thoracic aorta. Optimal visualization is obtained when a left anterior oblique position is employed. For left carotid arteriography within the skull, one

almost always must resort to direct puncture of the left common carotid artery.

COMPLICATIONS

In the initial stages of the examination, the patient will usually complain of muscular weakness in the hand or the whole upper limb. This rarely persists for more than 1 to

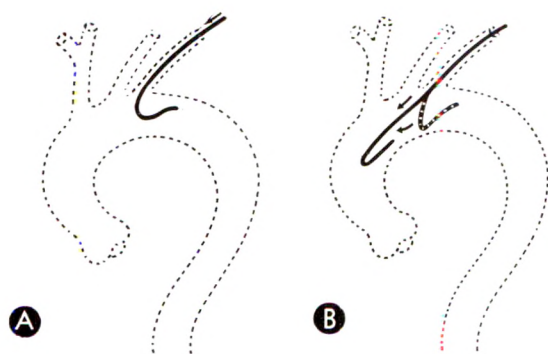


FIG. 26. Elongation and extreme uncoiling of the aortic arch by arteriosclerosis. Attempts to catheterize the descending thoracic aorta by the left axillary route sometimes result in a failure. Unless the catheter has a properly curved tip, it constantly hits against the lower wall of the aortic arch (A) and invariably goes into the ascending aorta (B) because of the displaced origin and abnormal direction of the left subclavian artery. Experience and more complicated maneuvers will usually overcome this difficulty.

2 hours and during a 3 year period no cases of permanent motor deficit have been observed. We believe that these transient effects are due to the local anesthetic agent or to the compression of the nerve branches either by edema or a small hematoma.

Temporary numbness caused by irritation or trauma of the brachial plexus and its branches is the rule in all cases. Usually, the fingers are affected in the radial, ulnar or median nerve distributions, sometimes the whole hand and the forearm, and rarely the shoulder. Complete disappearance of the numbness occurs within 1 to 2 hours after the examination. Axillary compression after completion of the procedure may often produce a temporary recrudescence of the discomfort. Occasionally, discrete numbness of one or more fingers persists. In 20 cases (4.2 per cent) slight alteration of the sensation of the pulp of the fingers persisted for 24 to 48 hours. Six other patients (1.3 per cent) had numbness of the tip of one finger for a few weeks without serious disability and with gradual return to normalcy. In 4 patients (0.8 per cent), the numbness was still present in one or many fingers 1 year after the examination.

In 8 cases (1.7 per cent), temporary disappearance or weakening of the brachial or radial pulse, probably as a result of arterial spasm, was observed for a few hours following axillary catheterization.

In 8 patients (1.7 per cent), the pulse was permanently absent and 3 of these (0.6 per cent) required axillary thrombectomy. The remaining 5 patients (1.1 per cent), presumably because of a well established collateral circulation, did not require surgery.

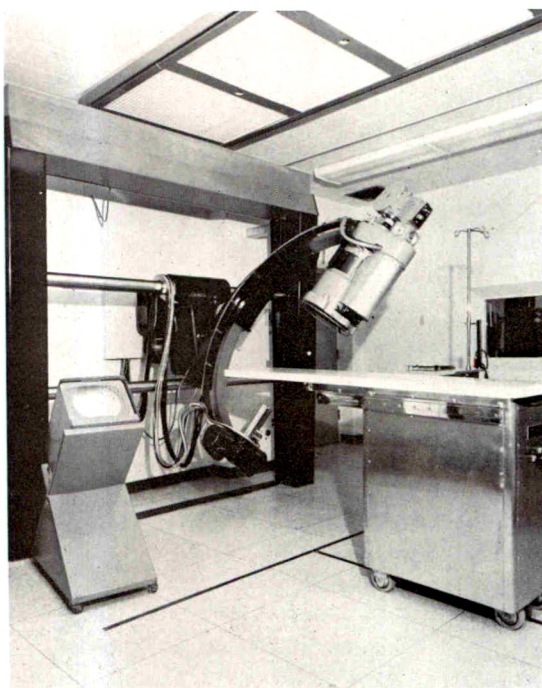


FIG. 27. Unit of vascular and cardiac catheterization as conceived at the Hôtel-Dieu Hospital of Montreal ("Saturn" apparatus). Televised radiocopy and radiocinematology may be performed in any plane, without having to modify the positioning of the patient during the examination. The roentgen-ray tube is placed at one extremity of a mobile arm shaped as a "C." At the opposite extremity are suspended the amplifier, videocamera and cinecamera (35 mm.). This complex can be moved in orbit or straight line in all planes around the table, by remote control. The transparent table top, at the level of abdomen, thorax and head, has no metal frame which may obstruct rays. Movements of the apparatus are controlled by an aseptic and removable push button panel, convenient to the operator. Television picture is recorded on video tape.

To minimize the risk of spasm or thrombosis, we usually inject a vasodilator by the intramuscular route prior to examination. Upon completion of the procedure, the same drug is slowly administered through the catheter just before its withdrawal. In this fashion, the injection is made a few centimeters proximal to the site of the puncture. All of the tubing used for these investigations has been coated with silicone.

Small hematomas and ecchymosis of the axilla, extending to the inner aspect of the arm, are frequently seen after percutaneous artery catheterization, but these are of no significance. Seven cases (1.5 per cent) were complicated by a larger hematoma, one of which (0.2 per cent) necessitated surgical drainage after suture of the small arterial puncture.

SUMMARY

In a series of 3,476 vascular studies performed over a period of 3 years, the author has employed percutaneous arterial catheterization in 1,149. Many of these cases were investigated by the femoral approach. When the femoral route failed or was impractical, percutaneous brachial or radial catheterization was sometimes attempted, but the results again were usually mediocre and the complications frequent.

To overcome these difficulties the author has adopted a new approach; *i.e.*, percutaneous puncture and catheterization of the axillary artery. Over a period of 3 years, 475 axillary artery punctures were performed, 79 of which were for opacification of the arteries of the upper limb by direct injection and 396 for introduction of a catheter for various types of selective arteriography.

The technique is briefly described and its applications enumerated. The limitations and specific obstacles to the use of the procedure are considered. A review of the material revealed the sites where certain arterial changes prevented the passage of the catheter in 46 cases (11.6 per cent). The infrequent complications of the method are

listed and certain difficulties of the examination which have become evident during the author's personal experience are discussed.

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PERCUTANEOUS RETROGRADE BRACHIAL ARTERIOGRAPHY*

A NONOPERATIVE, NONCATHETER TECHNIQUE

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RETROGRADE injection of contrast material into the brachial artery has been practiced in this institution for approximately 5 years. This procedure has been used primarily for roentgenographic evaluation and visualization of the intracranial vessels and, to a lesser extent, for the cervical carotid and vertebral arteries. Originally, the open cannula method first reported by Castellanos and Pereiras in 1959 was used exclusively.^{1,10,11} Since the introduction of the percutaneous method of arterial injection several years ago, this method has been employed in an increasing number of cases for visualization of the vertebral-basilar artery system on the left and the carotid and vertebral-basilar systems on the right side. At the present time, the open cannula method has been almost completely replaced except for small children.

The advantages of the percutaneous method are several. An initial operative procedure with its attendant, although minimal, anesthesia risk is avoided. An operating room and nurse assistance is not needed. The time required for the entire procedure is considerably reduced and the brachial artery is traumatized to a lesser degree. In addition, an operative closure of the vessel following the procedure is eliminated. The combination of right percutaneous brachial and left carotid arteriography at one sitting provides an almost complete or global angiogram, visualizing the bilateral carotid, right vertebral, and basilar systems. True 4 vessel arteriography may be accomplished also at one sitting by use of bilateral percutaneous brachial and left carotid arteriography.

METHOD

The necessary equipment consists of the usual angiographic tray, a No. 17 gauge thin-wall Courmand arterial needle, and a suitable automatic pressure injection machine. Usually, the pulse of the brachial artery is easily palpable in the antecubital fossa, even in rather obese patients. It has been a rare occurrence to be unable to perform the study because of inability to palpate the artery. During this consecutive series, in only 4 instances were attempts to puncture the brachial artery unsuccessful. (These were excluded from the series.) In one other case, puncture of the vessel was accomplished, but the return flow was unsatisfactory and the injection was not made. A successful puncture was done at the same site at a later time.

The procedure is performed under local anesthesia. Preoperative sedation consists of an appropriate barbiturate and atropine, usually combined with prochlorperazine. The arm to be injected is abducted and hyperextended at the elbow and supported in an appropriate manner. A small amount of local anesthetic is usually injected around the brachial artery as well as in the skin. The brachial artery is punctured at its most prominent point, in the upper one-half of the antecubital fossa over the prominence of the brachialis muscle. At this point the median nerve lies on the ulnar side of the vessel (Fig. 1 and 2). The needle is carefully threaded its entire length into the artery in a retrograde direction. Great care must be taken to avoid disruption of the intima or passage of the tip of the needle into a branch. A sphygmomanometer cuff is wrapped on the forearm and inflated to 300

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FIG. 1. The position of the brachial artery (solid line) is shown in relation to the median nerve (dotted line) as these structures enter the antecubital fossa. The sphygmomanometer cuff is seen in place on the forearm.

mm. mercury prior to the injection. Vascular compression by the cuff probably reduces the loss of contrast material distally, and prevents flooding of the hand with contrast material, which can be painful. Injection of approximately 45–50 cc. of 60 per cent renografin (30–35 cc. in children) is made utilizing the Taveras Automatic Injector at 525–625 lb./sq. inch pressure. The duration of injection is about 2 seconds. The operator should place the fingers of the left hand over the vessel above the needle during the injection in order to detect extravasation. The injection should be made only if arterial pulsation is noted in the connecting tube indicating that the needle still is within the lumen of the artery (Fig. 3). If extravasation is noted, the needle should be immediately and quickly removed to avoid vascular injury by subintimal injection of large amounts of contrast material. Starting during the injection, serial films are then exposed to ob-

tain complete arterial and venous phases in the anteroposterior and lateral projections simultaneously.

The roentgenographic technique used for recording cerebral arteriography is essentially unchanged since our previous reports.^{2,11,13} The Chamberlain biplane stereoscopic angiographic device is still being employed. This device provides rapid serial roentgenography in two planes. Motion of the tubes occurs between exposures for stereoscopy and each film of the series is a stereoscopic mate with its adjacent film.

Stereoscopy has distinct advantages over the ordinary methods for evaluation of vessel location and spatial relationships of vessels within the skull. In addition, the advantages of simultaneous biplane filming are readily recognized. Spasm or faulty injection can cause dissimilar filling on repeated injections, and accurate localization and correlation between anteroposterior

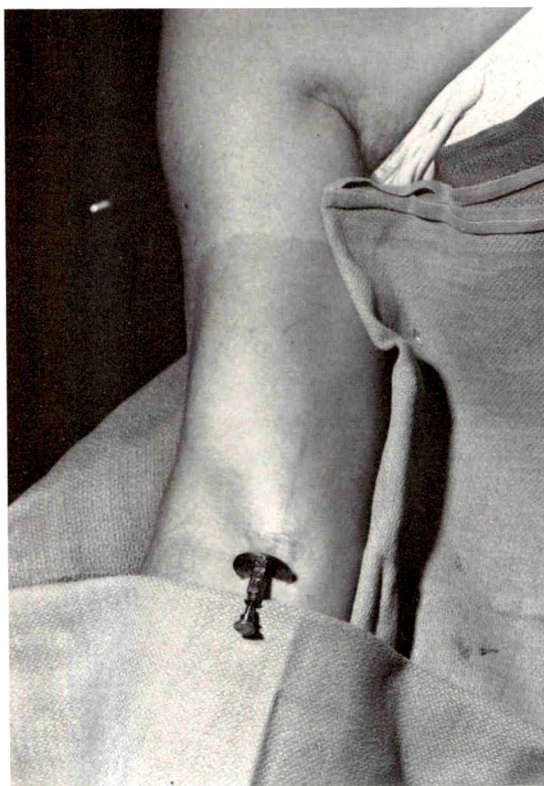


FIG. 2. The No. 17 gauge thin wall Courmand needle is shown in place after puncture of the brachial artery. The solid occluding stylet has been inserted.

and lateral projections may not be possible if multiple injections are needed.

In addition, the amount of contrast material needed for complete cerebral vascular evaluation is markedly reduced by the single injection technique. If 2 consecutive injections must be made, the connecting tubing should be disconnected at the needle and the solid stylet inserted to prevent clotting in the needle until reloading of the injection syringe can be accomplished.

As the needle is withdrawn, digital compression is made by the operator over the site of puncture for a full 5 minutes. Sufficient pressure is exerted to prevent hematoma formation, but not enough to obliterate the radial pulse. An ice bag is placed on the area for about 6 hours and frequent checks are made of vital signs, the area of puncture, and the arm for signs of hemorrhage or ischemia during the first day following the procedure.

In this series, 124 percutaneous brachial angiographies performed in 117 patients were reviewed. Of this group, 109 were right brachial injections; 68 of these were combined with left carotid arteriography at the same sitting. Left percutaneous brachial injection was performed in 15 cases. Contrast material reached the intracranial circulation in all cases of the left brachial injection, whereas in 4 instances with right brachial injection, no contrast material was identified in the neck or head, despite technically good injections and lack of clinical evidence of vascular occlusion. In one other case there was complete lack of filling

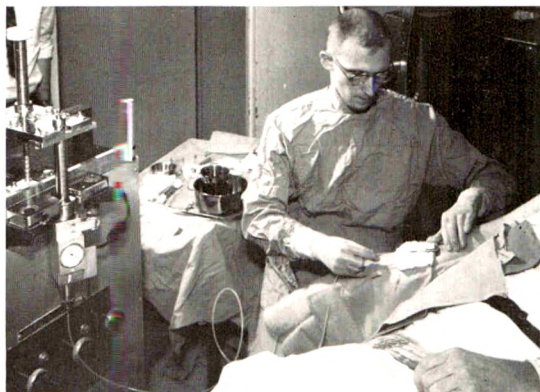


FIG. 3. The power injector (Taveras) is shown connected to the needle by the high pressure tubing. The pulsating reflux of blood can be seen by the operator in the tubing adjacent to the needle, indicating satisfactory placement of the needle.

of the carotid artery while the vertebral artery was well visualized. There was no clinical evidence of vascular occlusion in this patient. The absence of contrast in these cases is thought to be due to congenital variation in the origin of the subclavian artery.

RESULTS

In 105 right brachial angiograms in which contrast was identified in the neck or above, there was good visualization of the carotid artery and branches in 98 cases (93 per cent). In 2 cases (2 per cent) the major vessels only could be traced, and in 5 cases (5 per cent) visualization was unsatisfactory. In 120 right and left brachial injections, the vertebral artery was well visualized in 119 cases (99 per cent). In 105 right brachial injections, the basilar artery and branches were well visualized in 82 cases (79 per cent) (Table 1). Opacification of the basilar artery was variable but sufficient to trace the vessel in 11 cases (10 per cent), and unsatisfactory in 12 cases (11 per cent). In 15 left brachial injections, the basilar artery and branches were well visualized in 13 cases (87 per cent), variable in 1 case, and unsatisfactory in 1 case (this patient later proved to have occlusion of the basilar artery at autopsy).

It was found that visualization of the posterior cerebral arteries and other branches paralleled the adequacy of visual-

TABLE I

QUALITY OF VISUALIZATION OF MAJOR ARTERIES
IN 105 RIGHT BRACHIAL ARTERIOGRAMS

	Carotid Artery	Vertebral Artery	Basilar Artery
Good	98 (93%)	104 (99%)	82 (79%)
Usable*	2 (2%)	—	11 (10%)
Unsatisfactory**	5 (5%)	1 (1%)	12 (11%)

* Visualization variable, but sufficient to trace the course of the vessel and for gross lesions.

** Visualization absent or not sufficient for interpretation.

TABLE II

VISUALIZATION OF THE POSTERIOR CEREBRAL ARTERIES IN 120 RIGHT AND LEFT BRACHIAL ARTERIOGRAMS

Quality of Basilar Artery	Posterior Cerebral Arteries Seen						Carotid Origin
	Both	Left Only	Right Only	R better than L	L better than R	None	
Good	73 (61%)	2 (2%)	4 (3%)	9 (7%)	9 (7%)	—	—
Usable	5 (4%)	—	1 (1%)	2 (2%)	—	2 (2%)	—
Unsatisfactory	1 (1%)	—	—	2 (2%)	—	6 (5%)	4 (3%)

ization of the basilar artery. There was no apparent selectivity of filling of the posterior cerebral arteries from either side, both sides being filled in about two-thirds of cases (Table II).

There was opacification of the left carot-

id vessels intracranially from below in 15 (14 per cent) of the right brachial studies.

Brachial arteriography was performed 47 times in 42 patients with spontaneous subarachnoid hemorrhage. Of these studies, 31 (66 per cent) were combined right brachial and left carotid arteriography. A diagnosis of aneurysm was made in 27 of these patients (Table III). Two patients were found to have arteriovenous malformations which had not bled.

Vascular occlusions were found in 4 patients, and vessel irregularities compatible with arteriosclerosis were found in 15 patients.

Twenty studies were performed in patients with clinical diagnoses of posterior fossa lesions other than vascular lesions. Of these studies, 8 were done with the clinical diagnosis of suspected cerebello-pontine-angle (CPA) lesion. In this total group, 2 patients were later found to have CPA tumors—neither was diagnosed by the angiogram. Two other patients were found to have cerebellar neoplasms which were suspected by angiographic findings (1 only in retrospect).

Supratentorial space taking lesions were diagnosed in 10 patients in this series—4 tumors, 1 cerebral edema, 3 subdural hematomas, and 2 intracerebral hematomas.

A defect due to a neurofibroma of the sixth cervical nerve root was demonstrated on a left brachial angiogram.

Four patients had bilateral brachial injections and 3 patients had repeat brachial injections during the same hospitalization.

TABLE III

LESIONS REVEALED BY BRACHIAL ARTERIOGRAPHY IN 120 PATIENTS

<i>Cerebral Aneurysms</i> —27	
Anterior communicating	6
Anterior cerebral	2
Middle cerebral	2
Posterior communicating	10
Basilar artery	4
Vertebral artery	1
Multiple	2
<i>Arteriovenous Malformations</i> —2	
Supratentorial	1
Infratentorial	1
<i>Vascular Occlusive Disease</i> —19	
Occlusion of carotid artery, extracranial	3
Occlusion of basilar artery	1
Vessel irregularity or partial block*	15
<i>Space Taking Lesions</i> —12	
Supratentorial	
Tumor	4
Cerebral edema	1
Subdural hematoma	3
Intracerebral hematoma	2
Infratentorial tumor	2
<i>Spinal Lesions</i> —1	
Neurofibroma	1

* Vessel changes in regularity or caliber interpreted to be on an arteriosclerotic basis.

One of these patients had bilateral brachial arteriography for diagnosis and evaluation of a vertebral artery aneurysm; after clipping of the vertebral artery intracranially below the aneurysm, repeat left brachial arteriography was performed and revealed nonfilling of the aneurysm.

MORBIDITY AND MORTALITY

There was a remarkable lack of immediate effects from this procedure. The discomfort on injection is perhaps slightly greater than that experienced during carotid arteriography, but the puncture of the vessel and subsequent discomfort are much less distressing than with the carotid procedure.

One death occurred 10 hours following right brachial arteriography. This patient had been admitted in a stuporous condition with the clinical diagnosis of hypertension with severe cerebrovascular hemorrhage. The angiogram showed no evidence of a space-taking lesion. Autopsy revealed a 1.5 cm. hematoma in the right basal ganglia, intraventricular hemorrhage, and an area of hemorrhage in the pons measuring 2.0 cm. It was considered that the arteriography had no direct causative effect in the mortality.

There was no incidence of focal motor weakness. One patient had a brief left sided focal seizure.

There were miscellaneous reactions: 1 patient had a severe spasm of coughing following the injection, 2 patients manifested transient neurogenic shock upon puncture of the artery, 1 patient developed a transient urticarial reaction on the face immediately after the injection, and 1 patient developed a body rash 1 day following the study, which gradually subsided with antihistamine treatment.

Morbidity of a local nature was minimal. One patient manifested slight numbness and tingling in the right thumb following a right brachial injection. This numbness subsided within a few days. The postprocedure course of 114 patients was reviewed. There were no specific remarks concerning pain in the arm, hematoma, or signs of is-

chemia in 62 cases. In 45 cases records specifically showed that these complications were absent. Significant pain at the site of puncture was present in only 2 patients and was caused by an extravasation of contrast material. Significant hematoma formation occurred in only 2 cases. Pain or coldness in the forearm was noted in 3 cases; in only 1 of these were the symptoms persistent at the time of discharge. This latter patient was the only case in which there was a loss of the radial pulse with no return. In 38 cases the radial pulse was recorded as being intact following the procedure. In 71 other cases no note was made about the presence or absence of pulse; however, the unrecorded observations of the staff have been that few, if any, patients have had reduction or loss of the pulse. Total extravasation of the contrast material occurred in 1 case with subsequent redness and slight swelling in the upper arm. There were no signs of subsequent vascular embarrassment or other problems in that extremity. Partial extravasation occurred in 1 other case, with no effect on the radial pulse or subsequent disability.

DISCUSSION

It is our purpose to review our experience with percutaneous retrograde brachial arteriography and to recommend early, complete (global) intracranial vascular visualization in patients manifesting spontaneous subarachnoid hemorrhage. We believe, with many others, that arteriography is indicated to establish a diagnosis in these cases as early as is practicable or when the patient's condition is clinically stable. This may vary from 2 hours to 5 days—generally 24 to 72 hours.

Prior to the use of retrograde brachial injection, cases manifesting subarachnoid hemorrhage have been studied by means of bilateral carotid arteriography. In occasional cases with presenting signs or symptoms suggesting a posterior fossa lesion, visualization of the vertebral-basilar system has been accomplished by direct vertebral arteriography. Percutaneous vertebral artery puncture is not only difficult but gen-

erally very painful. It has been estimated that only 70–80 per cent are successful, even in experienced hands. The procedure has many inherent hazards imposed by the normal anatomy. The open retrograde brachial injection by cannula, and vessel injection by catheter techniques are relatively time consuming compared to the percutaneous technique, although they may be necessary if percutaneous puncture and injection cannot be performed for some reason.

The availability of reliable automatic power injection machines has made possible the use of the relatively small bore needles which are required in order to accomplish an easy and nontraumatic percutaneous puncture of the brachial artery. It previously has not been possible to inject by hand power a bolus of contrast material through such a small bore needle in the short period of time (about $1\frac{1}{2}$ to 3 seconds) necessary to provide retrograde filling of the subclavian and innominate arteries sufficient to adequately fill the intracranial vessels. It has been possible in our experience to accomplish the goal of almost complete intracranial arteriography (excepting left vertebral) by means of two needle

punctures (right brachial and left carotid) in a total time of 20 to 30 minutes.

ILLUSTRATIVE CASES

The following cases illustrate the use of this technique.

CASE I. I.A., a 46 year old chronic hypertensive female, was admitted with spontaneous subarachnoid hemorrhage manifesting no focal neurologic signs. Right brachial and left carotid angiograms showed bilateral aneurysms in the right anterior cerebral, right and left posterior communicating and left posterior cerebral artery (Fig. 4, *A* and *B*; and 5, *A* and *B*). Post-mortem examination later revealed a total of 7 aneurysms.

Comment. This case re-emphasizes the well known possible multiplicity of aneurysms.

CASE II. A. W., a 46 year old female, was admitted with a second subarachnoid hemorrhage, exhibiting no localizing signs. Right brachial and left carotid arteriography was performed revealing an aneurysm of the basilar artery bifurcation. This patient was discharged without surgical treatment and was shortly thereafter readmitted with a third and fatal hemorrhage (Fig. 6, *A* and *B*).

Comment. This case demonstrates how

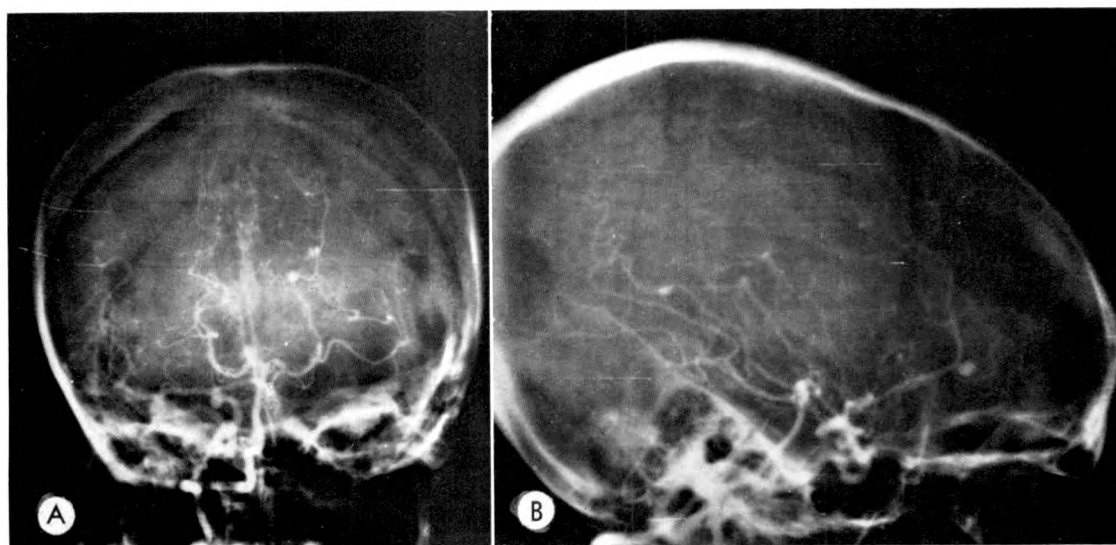


FIG. 4. Case I. (*A*) Anteroposterior projection of the arterial phase of right retrograde brachial arteriography shows aneurysms of the right posterior communicating artery, right anterior cerebral artery and left posterior cerebral artery. (*B*) Simultaneous lateral projection shows similar findings.

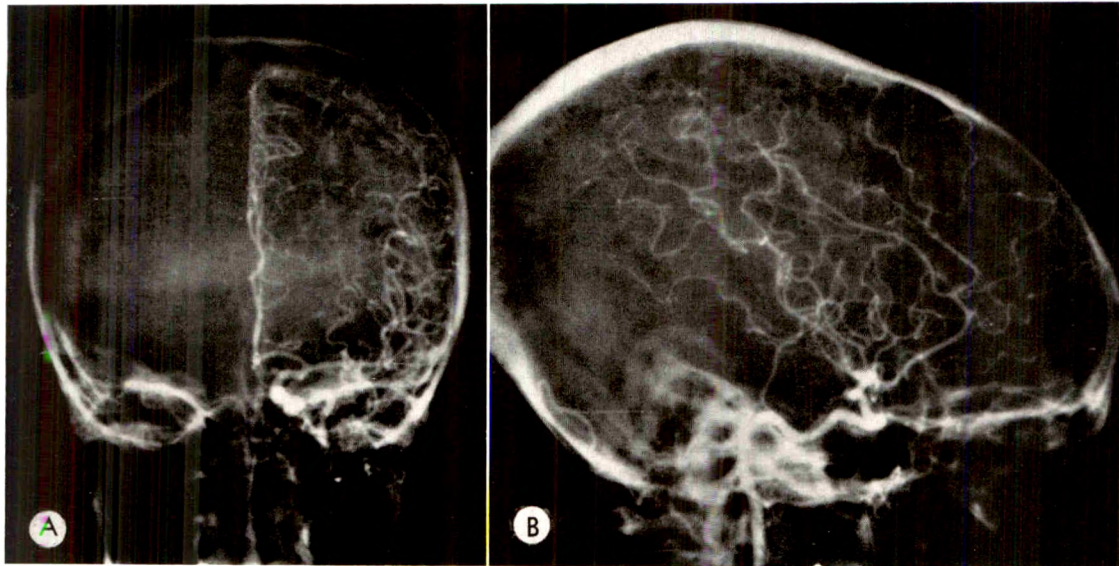


FIG. 5. Case 1. (A) Anteroposterior projection of left carotid arteriography shows a small aneurysm of the left posterior communicating artery. (B) Simultaneous lateral projection again shows the aneurysm.

the basilar artery aneurysms may bleed and not produce suggestive localizing signs. As more of these lesions are found, perhaps methods of direct management will be perfected.

CASE III. L. W., a 50 year old female, was

admitted with spontaneous subarachnoid hemorrhage and transient weakness of the left side. Bilateral brachial and left carotid arteriography revealed a large aneurysm of the right vertebral artery just above the posterior inferior cerebellar artery (Fig. 7, A and B; and 8, A and B). A suboccipital craniotomy was performed and

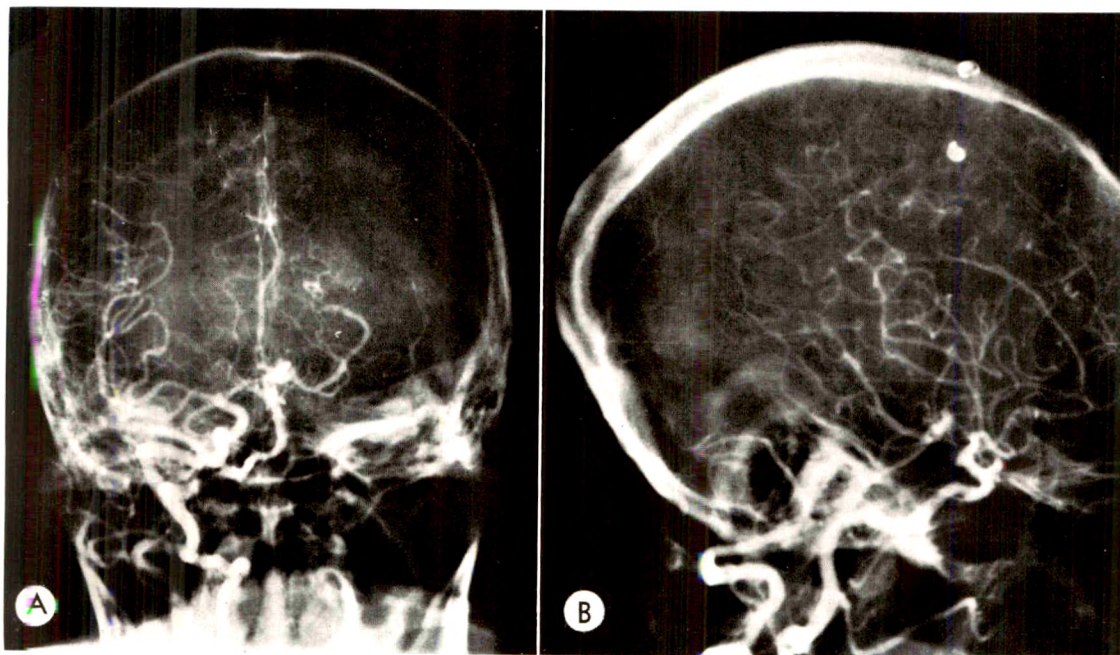


FIG. 6. Case II. (A) Anteroposterior and (B) lateral views showing the arterial phase of right retrograde brachial arteriography. A saccular aneurysm can be seen at the top of the basilar artery.

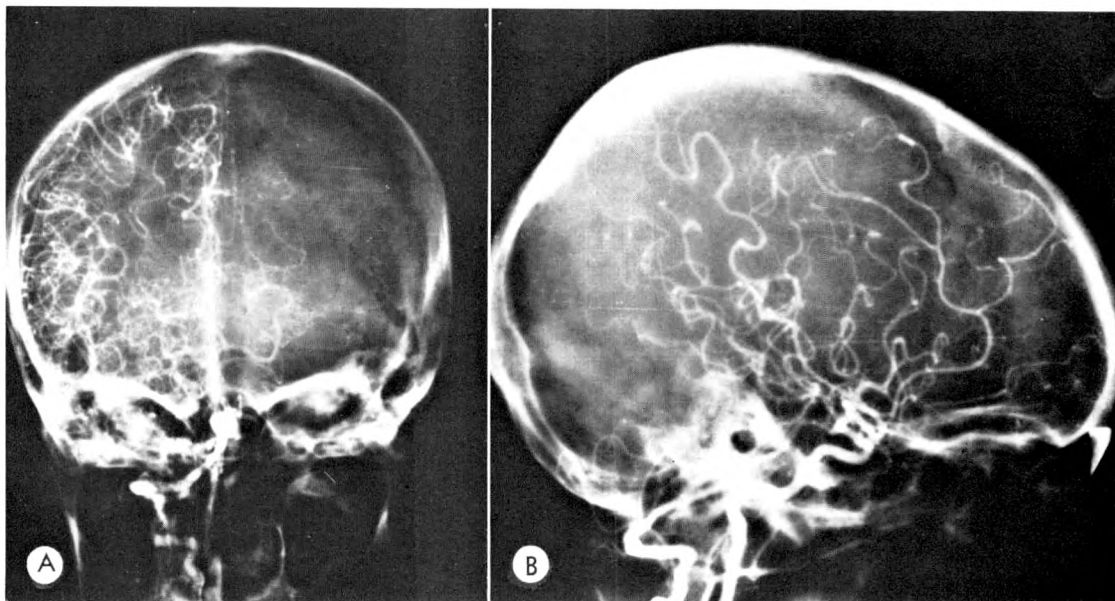


FIG. 7. Case III. Simultaneous (A) anteroposterior and (B) lateral views of the arterial phase of right retrograde brachial arteriography show a large aneurysm of the right vertebral artery just above the origin of the posterior inferior cerebellar artery.

the right vertebral artery was clipped below the aneurysm. The patient has done well post-operatively.

Comment. This case demonstrates the diagnosis and evaluation of a posterior

fossa aneurysm by bilateral brachial arteriography and the surgical treatment of the lesion.

CASE IV. J.S., a 40 year old male, was readmitted for evaluation of recurrent headaches.

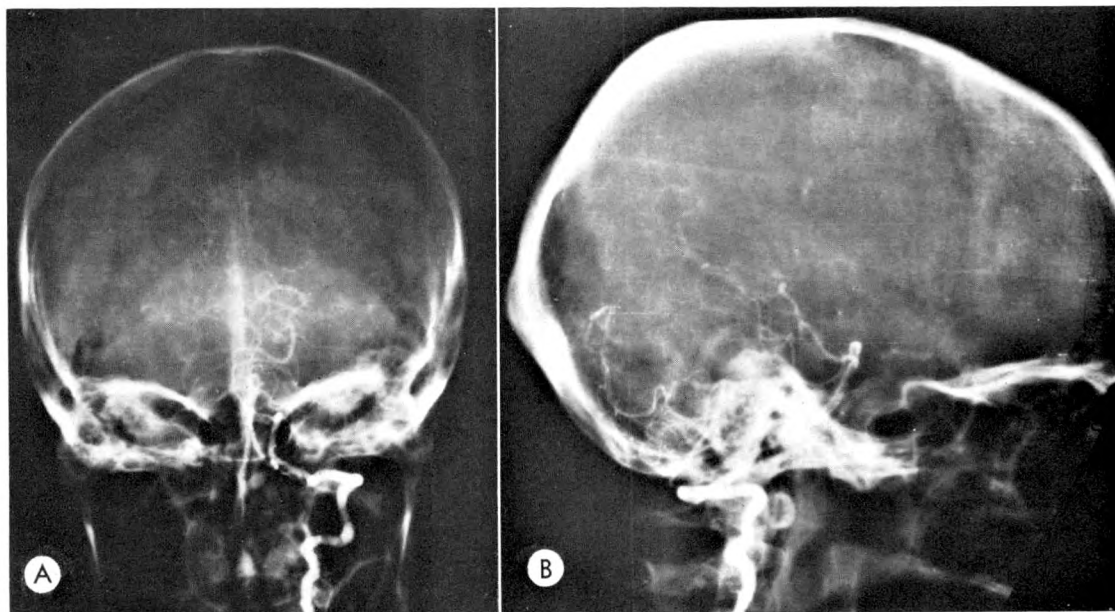


FIG. 8. Case III. (A and B) Left retrograde brachial arteriograms show no retrograde filling of the aneurysm of the right vertebral artery.

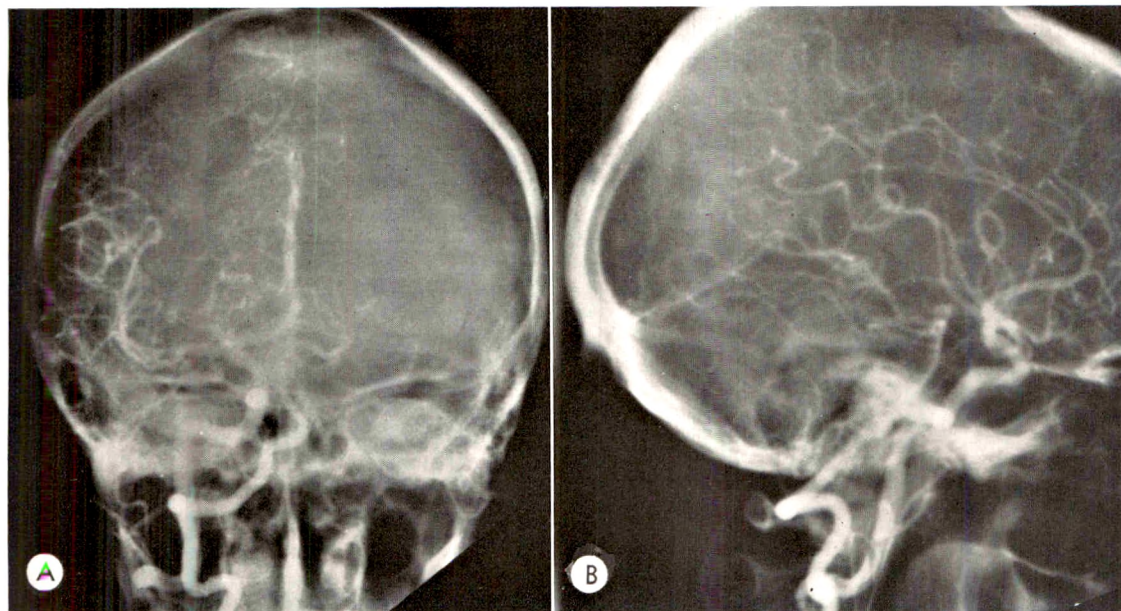


FIG. 9. Case IV. (A) Anteroposterior view shows irregular angiomatic vessels arising from the right posterior cerebral artery. (B) The lateral view shows these same vessels in the region of the quadrigeminal plate with shunting of opacified blood into the vein of Galen and early filling of the straight sinus.

This patient was studied 2 years previously for spontaneous subarachnoid hemorrhage associated with nystagmus, paralysis of upward gaze, and loss of pain and temperature sensation in the left arm and face. An open right brachial arteriography was performed at that time using multiple injections and single roentgenographic films. A questionable abnormality was noted above the region of the basilar artery but no definite diagnosis could be established angiographically. Because of increasing headaches, the patient was restudied by means of percutaneous right brachial arteriography. This revealed an arteriovenous malformation in the region of the quadrigeminal plate (Fig. 9, A and B).

Comment. Of interest in this case is the fact that the percutaneous study was easily performed in the same extremity where the open brachial study had been done previously. The value of the serial biplane stereoscopic studies was readily evident.

CONCLUSIONS

Percutaneous retrograde brachial arteriography has provided us with a reliable, safe method for the rapid and complete

evaluation of the right carotid as well as the posterior fossa circulation. When right brachial injection is combined with percutaneous left carotid arteriography, almost complete intracranial vascular visualization is obtained. In this way, it is possible to quickly establish a diagnosis in cases of subarachnoid hemorrhage with a minimum of stress to the patient. The desirability of complete vascular study in cases of spontaneous subarachnoid hemorrhage is obvious. Multiple berry aneurysms are not infrequent, and the pattern of vascular supply is necessary knowledge if surgical treatment is undertaken in cases with arteriovenous malformation. Note should be made of the rather high incidence of aneurysms in the posterior fossa circulation in this group of patients studied for subarachnoid hemorrhage (15 per cent of aneurysms found). Percutaneous brachial arteriography is more easily performed than the open procedure, and may be accomplished in less than one-third the time. Visualization is comparable by either technique, although the percutaneous method may have a slight advantage of greater reliability in

obtaining satisfactory studies. A lesser incidence (14 per cent) of cross filling of the left carotid artery occurred with right brachial injection in this group when compared with that previously reported (over 20 per cent) in the open technique series.¹¹ This is explained by the larger number of children included in the open series. Only 4 children were in the present series (ages 7-9 years), whereas there were 15 children (ages 10 or less) in the open series.

In cases where extensive arterial disease is present, the danger of complications attending direct carotid puncture are much increased. The use of right brachial injection obviates the necessity of direct puncture on that side. Brachial arteriography is preferable in patients who have mass lesions in the neck and in patients with tracheostomy.

Because of the number of studies in which there was unsatisfactory visualization of vessels—in particular the basilar artery—correlation was made with the clinical picture in order to attempt to determine whether this relative lack of filling was significant. In 5 cases of unsatisfactory visualization of the *carotid* artery, there was clinical evidence to suspect severe vessel disease in 3 cases. In the 1 case of unsatisfactory visualization of the *vertebral* artery, there were no referable clinical signs or symptoms. In 12 cases of unsatisfactory visualization of the basilar artery, only 3 cases manifested symptoms referable to disease of this vessel. In 8 cases, there were no symptoms suggestive of basilar artery disease; 1 case was doubtful. These data suggest that poor visualization of the vertebral or carotid arteries should be considered to be more significant than poor visualization of the basilar artery. The latter could be explained on a technical basis due to variations in mixture of contrast material and rate of flow from the opposite vertebral artery. Included in those cases in which there was unsatisfactory visualization of the basilar artery were 3 cases in which excellent filling of the vertebral and posterior inferior cerebellar (PIC)

artery was obtained, but no contrast material was seen in the vertebral or basilar artery above the PIC artery. In none of these were there symptoms to suggest vascular insufficiency in this region. Morris⁹ has previously reported a group of patients showing this abnormality in which hypogenesis of the vertebral artery ("non-union of the vertebral arteries") exists due to failure of development of junction with the opposite vertebral artery. The basilar artery is supplied by the other, larger, vertebral artery.

One other case was encountered subsequently with this angiographic finding which came to surgical exploration because of pneumoencephalographic evidence of a cerebello-pontine-angle mass lesion. A large clotted aneurysm was found arising from the vertebral artery at the level of the PIC artery.

As described previously,¹¹ proper positioning will allow visualization of the cervical portions of the right carotid and vertebral arteries. Opacification of the proximal innominate and left subclavian arteries has been variable due to mixture with larger volumes of unopacified blood.

Although the diagnosis of posterior fossa space-taking lesions is difficult by arteriography and certainly less reliable than air study, its use should be encouraged, particularly where stereoscopy is available. As technique and ability to interpret these studies inevitably will improve, it may be possible to establish firm enough diagnoses by arteriography to allow us to avoid the more dangerous air study as we now do in many cases of supratentorial lesions.

SUMMARY

1. Percutaneous retrograde brachial arteriography, a nonoperative, noncatheter technique, has been demonstrated to be a rapid, safe, reliable, relatively nontraumatic procedure for positive contrast visualization of the intracranial and cervical vasculature.

2. Specific emphasis is placed on the routine use of combined right brachial and

left carotid arteriography in the patient with spontaneous subarachnoid hemorrhage. Complete intracranial vascular visualization is considered necessary in these cases. A rather high incidence of posterior fossa aneurysms has been found in this series.

3. The usefulness of percutaneous retrograde brachial arteriography in the investigation of vascular occlusive disease and supratentorial space taking lesion has been pointed out.

4. Correlation of clinical and arteriographic findings has demonstrated that poor opacification of the basilar artery and branches is more likely to be on a technical basis and not as clinically significant as poor opacification in the carotid system.

5. Increased use of brachial arteriography in the diagnosis of posterior fossa space taking lesions is urged.

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GUIDED ANGIOGRAPHY*

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SELECTIVE vascular catheterization followed by rapid delivery of the largest possible concentration of radiopaque material is the ideal method for optimal visualization of any vascular territory. Since 1962, we have developed instrumentation which facilitates cardiac and vascular cath-

eterization techniques. We have called this new approach "guided angiography."

TECHNIQUE

(1) A special two-piece cannula is used to puncture the vessel. It consists of an inner perforating, solid trocar and an outer cyl-

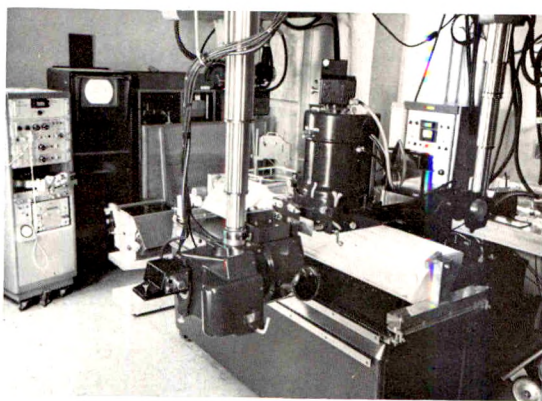


FIG. 1. Room for vascular studies: biplane cine unit (two 9 inch Picker image intensifiers) with television monitor; roll film changer; rotating cradle; recording device; DC defibrillator; apparatus for external cardiac massage; automatic pressure injector; home-made movable lead barriers.



FIG. 2. Instrumentation for catheterization studies.

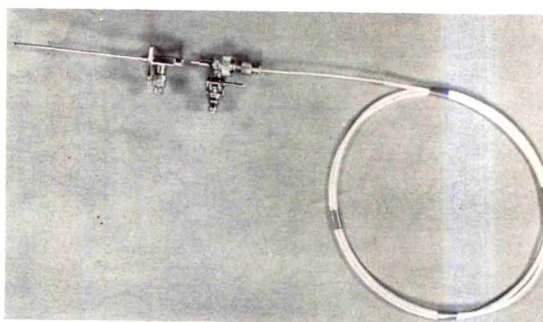


FIG. 3. Two-piece cannula, plastic-coated guide housed in a hollow, transparent guide receiver, and diaphragm-valve unit.

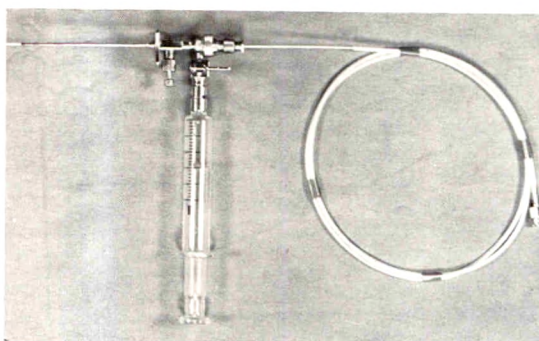


FIG. 4. Diaphragm-valve unit attached to cannula. The guide has been advanced.

* Presented at the Sixty-fifth Annual Meeting of the American Roentgen Ray Society, Minneapolis, Minnesota, September 29-October 2, 1964. A Scientific Exhibit on the same subject was awarded The Gold Medal of the Society.

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‡ Research Consultant.

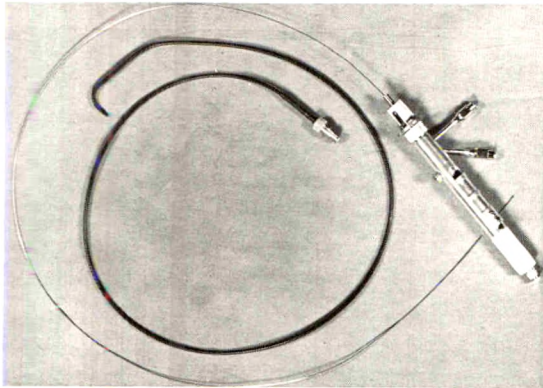


FIG. 5. Special catheter with deflector-rotator assembly.

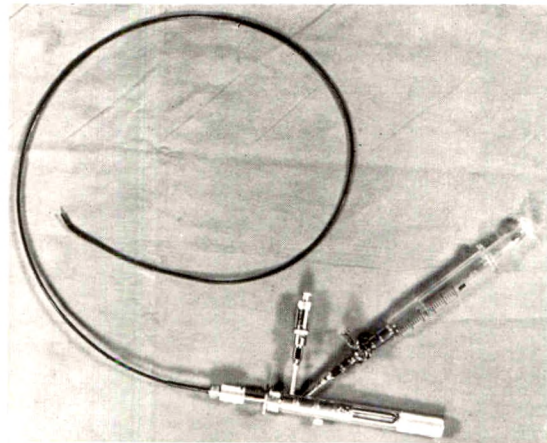


FIG. 6. Deflector-rotator assembly attached to the catheter.

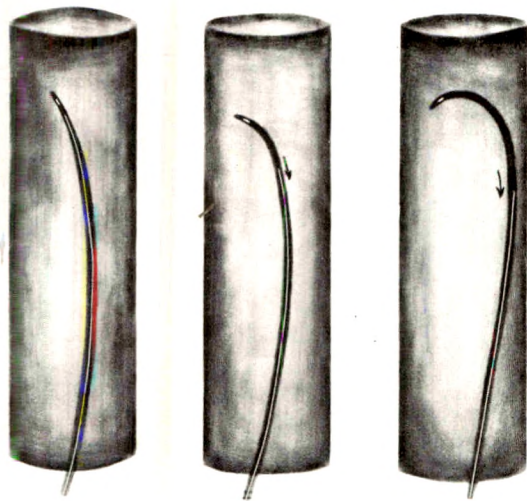


FIG. 7. Diagrammatic representation of controllable tip deflection. The metallic stylet straightens the distal curve of the catheter when advanced and the curve re-forms when the stylet is retracted.

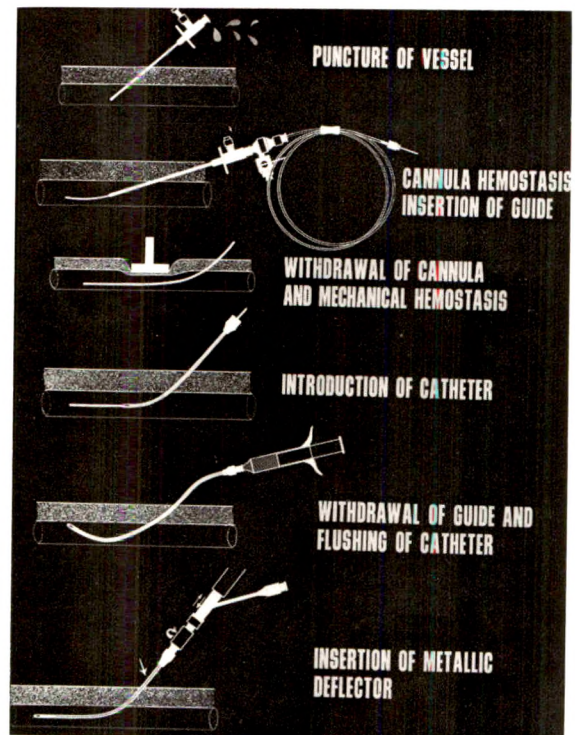


FIG. 8. Different steps in the percutaneous catheterization technique for guided angiography.

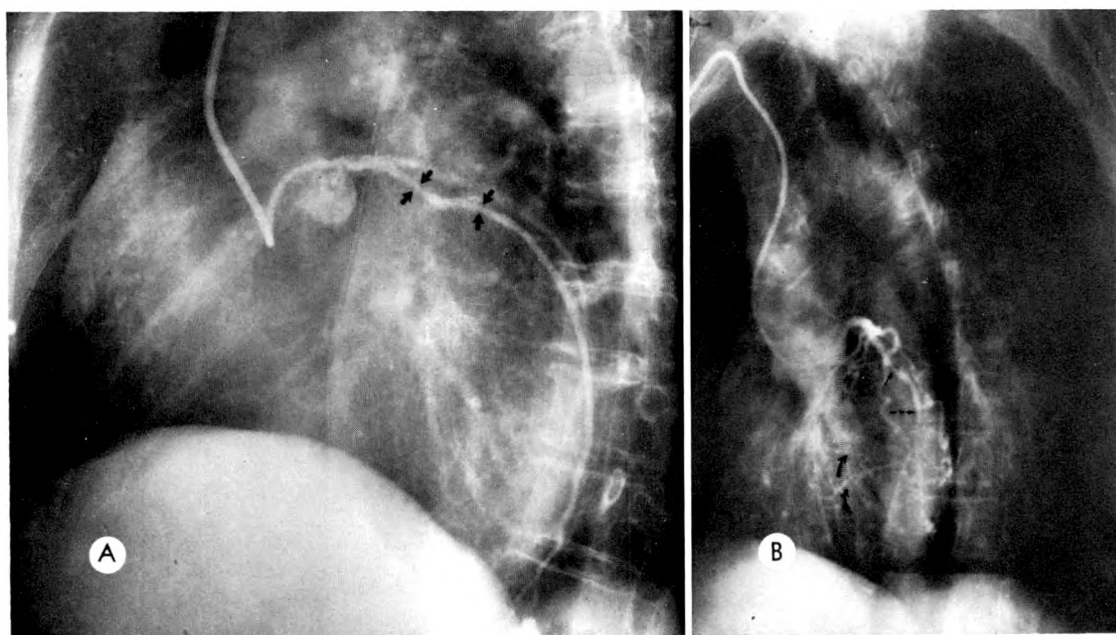


FIG. 9. (*A* and *B*) Selective left coronary arteriography. (*A*) Magnification technique. Note complete occlusion at the origin of the anterior descending division of the left coronary artery and arteriosclerotic narrowings of the circumflex (arrows). *B* was made by using the film changer. Note extensive arteriosclerotic changes and homocollateral circulation (arrows).

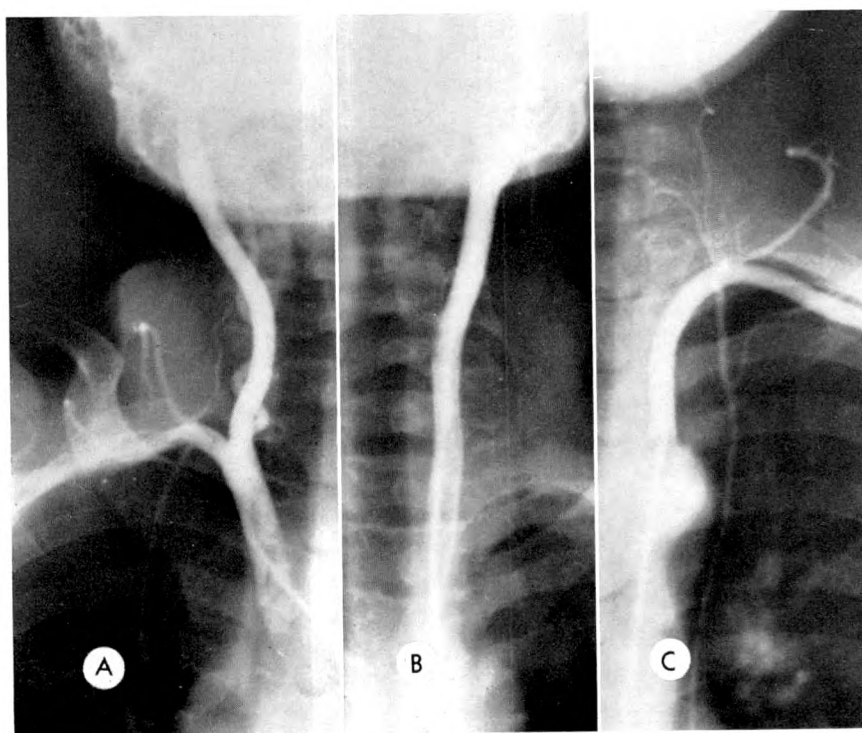


FIG. 10. (*A*, *B* and *C*) Selective innominate, left carotid and left subclavian arteriograms. Normal study.

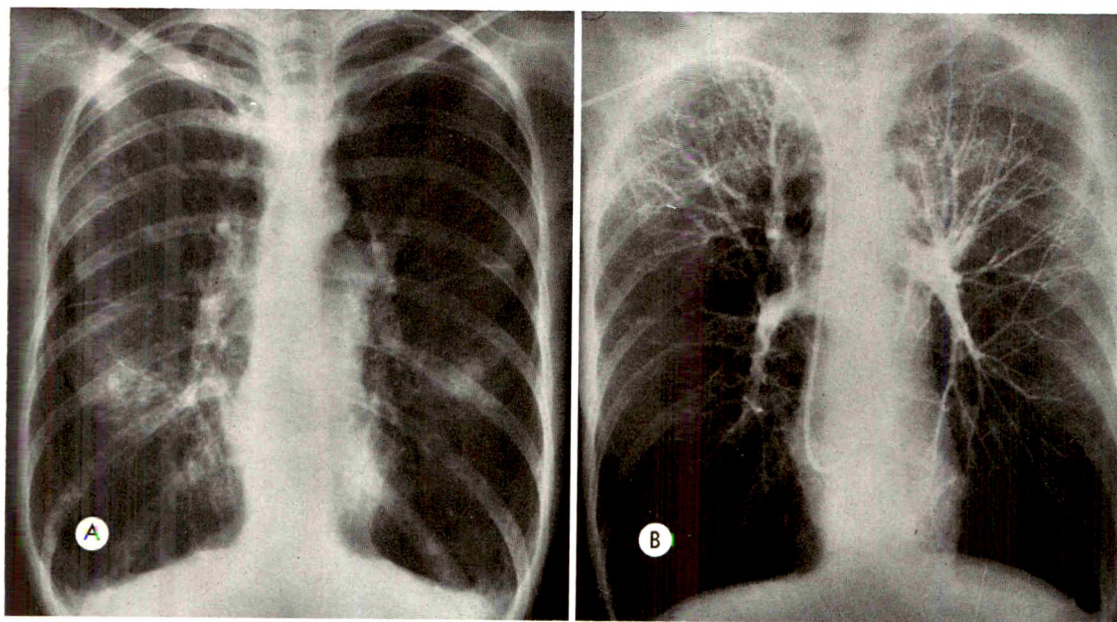


FIG. 11. (A and B) Pulmonary angiograms. (A) Diffuse emphysematous changes with area in the left upper lobe showing no vascular markings. (B) Selective pulmonary arteriogram showing the characteristic "winter-tree" pattern of diffuse pulmonary emphysema. Unexpected, numerous vessels are noted in the left upper lobe. The peripheral ischemia of the left upper lobe is due to the presence of a large bulla arising from the superior segment of the left lower lobe.

indrical, blunt cannula. Immediate hemostasis is accomplished by pressing a button that obturates the inner cannula. (2) A plastic-coated guide, housed in (3) a hollow, transparent guide receiver is inserted through the cannula. When the guide fits loosely in the cannula, (4) a diaphragm-valve unit which impedes blood loss during advancement of the guide is attached to the cannula. This diaphragm-valve unit has a side outlet which permits withdrawal of blood, flushing, and infusion of opaque medium without removing the guide. If there is impedance to the advancement of the guide, the anatomy of the catheterized vessel can be determined by the injection of contrast material.

After insertion of the guide, the cannula is withdrawn and (5) a special catheter is advanced over the guide. During this maneuver, (6) a mechanical compressor is employed to secure hemostasis. These special catheters have a soft, flexible, tapered, radiopaque tip, a relatively semirigid body which permits maximum rotational ability

without "whipping," and a tip with a wide range of controllable deflection. The catheters have two preformed curves. The distal (primary) one is hook-shaped; the proximal (secondary) one is broadarched. Interplay between the two curves adapts the catheter to vessels of different sizes. The catheters vary in length and caliber; both are important factors which influence the flow-rate of contrast medium.²

Following the introduction of the catheter, the guide is withdrawn and the catheter is flushed with heparinized solution. (7) A deflector-rotator assembly is then attached. A metallic stylet of the assembly straightens the distal curve of the catheter when advanced and the curve re-forms when the stylet is retracted.¹ When the catheter tip is in the desired vessel, the metallic deflector can be withdrawn or left in place. There are two outlets in the external portion of the deflector assembly. (8) A modified one-way valve is attached to one of the outlets. One outlet permits continuous monitoring of pressure and infusion of par-

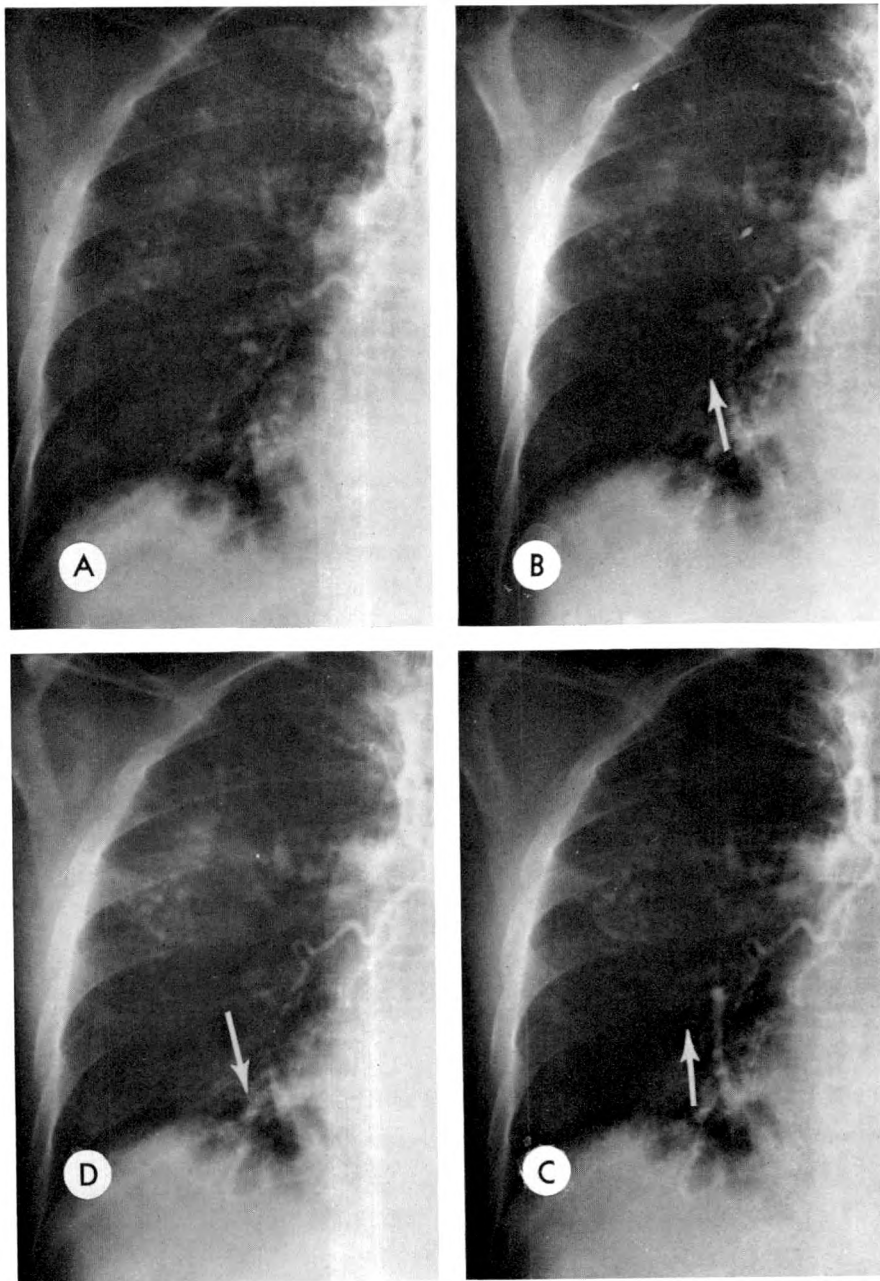


FIG. 12. (A-D) Bronchial arteriograms. Unresolved pneumonia in the right lower lobe. Note marked dilatation of the right bronchial arteries and opacification of the right lower branch of the pulmonary artery which fills from below and empties from above (arrow). This is due to reversal of flow in the pulmonary artery from precapillary anastomosis between the bronchial artery and the pulmonary artery.

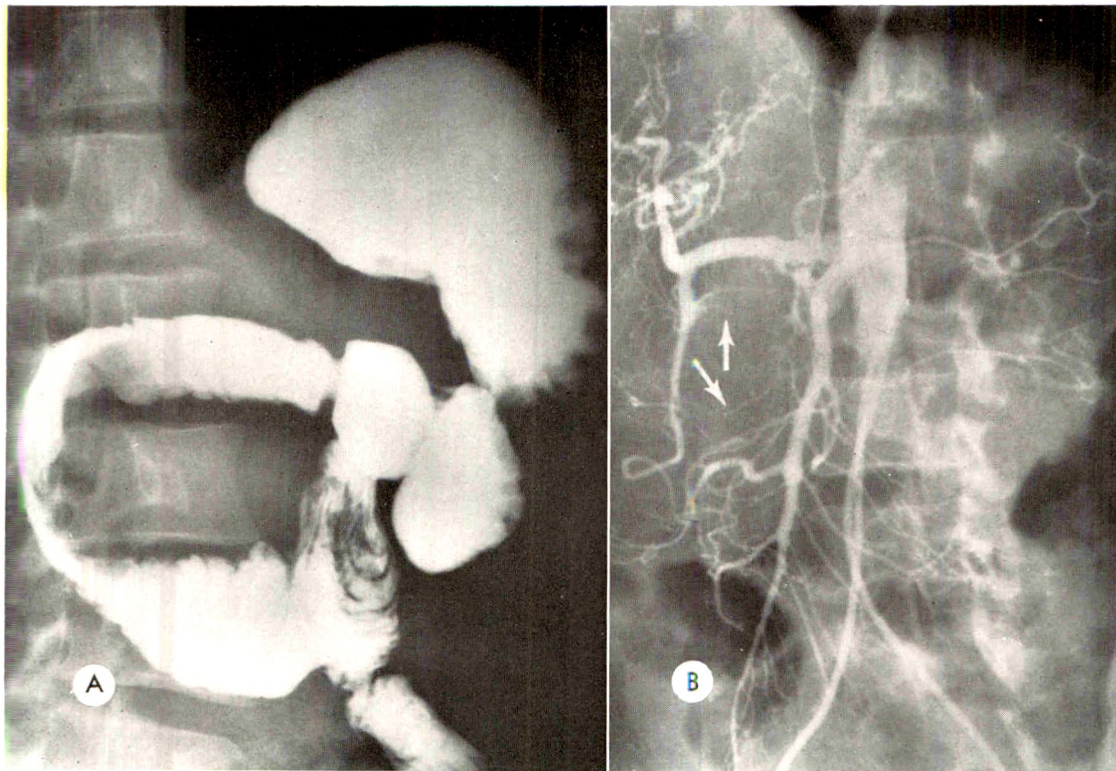


FIG. 13. (A and B) Pancreatic carcinoma. (A) Widening of the duodenal loop without invasion. (B) Simultaneous celiac and superior mesenteric angiogram showing displacement of vessels and fine peripheral tumor vessels (arrows).

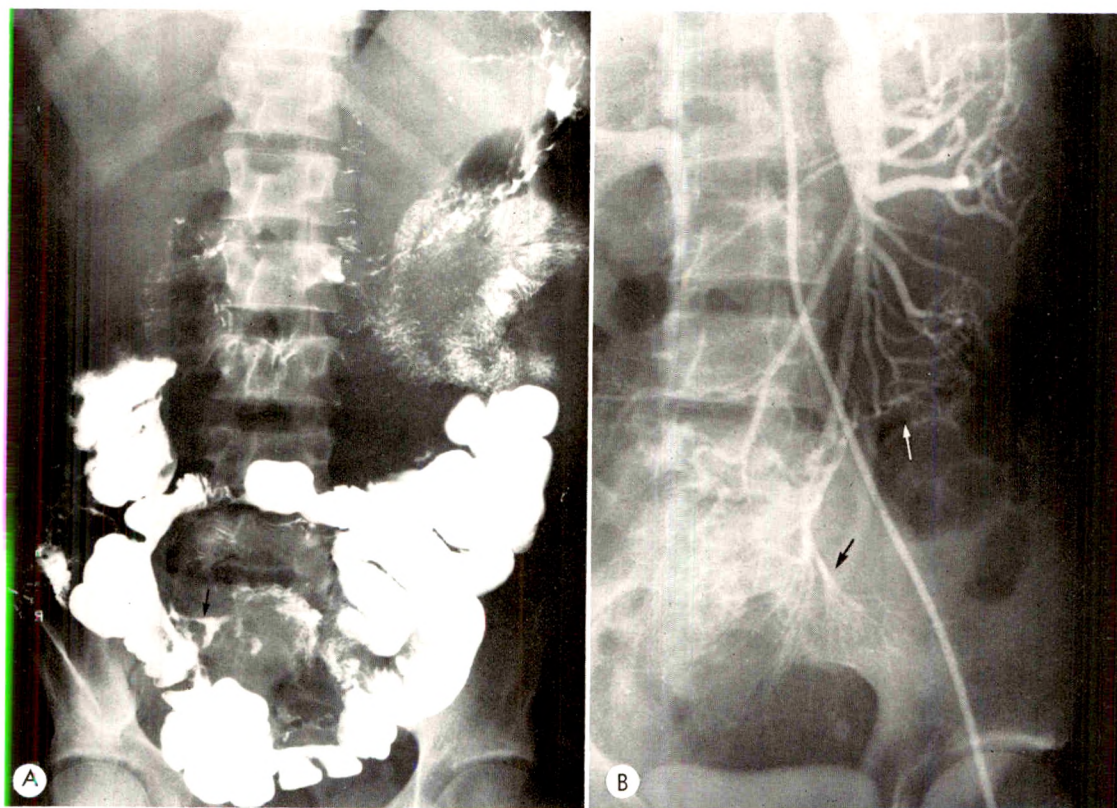


FIG. 14. (A and B) Regional enteritis. (A) Extensive small bowel changes, fistulous tracts (arrow) and mass displacing ileal loops. (B) Slight ileal hypervascularity. Compression of ileal vessels (arrows) from mesenteric mass which proved to be an abscess.

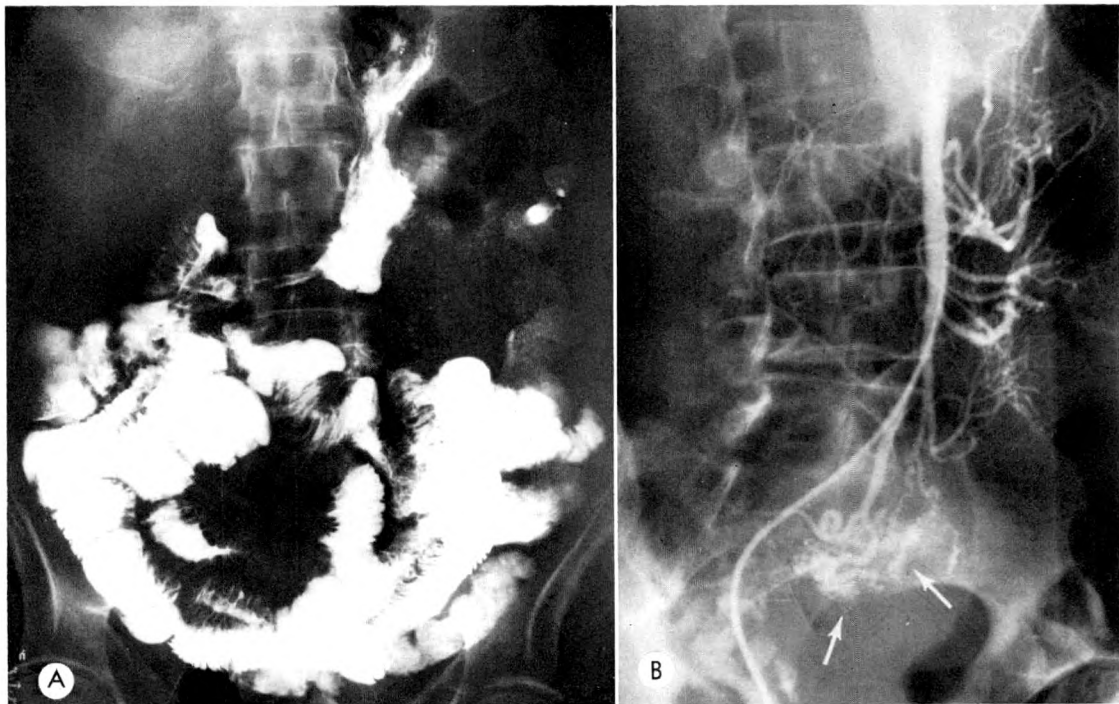


FIG. 15. (*A* and *B*) Malignant carcinoid. (*A*) Extensive small bowel changes simulating regional enteritis. (*B*) Selective superior mesenteric arteriogram showing ileal tumor vascularity (arrows). Edematous folds were secondary to lymphatic obstruction from a malignant carcinoid arising from the terminal ileum.

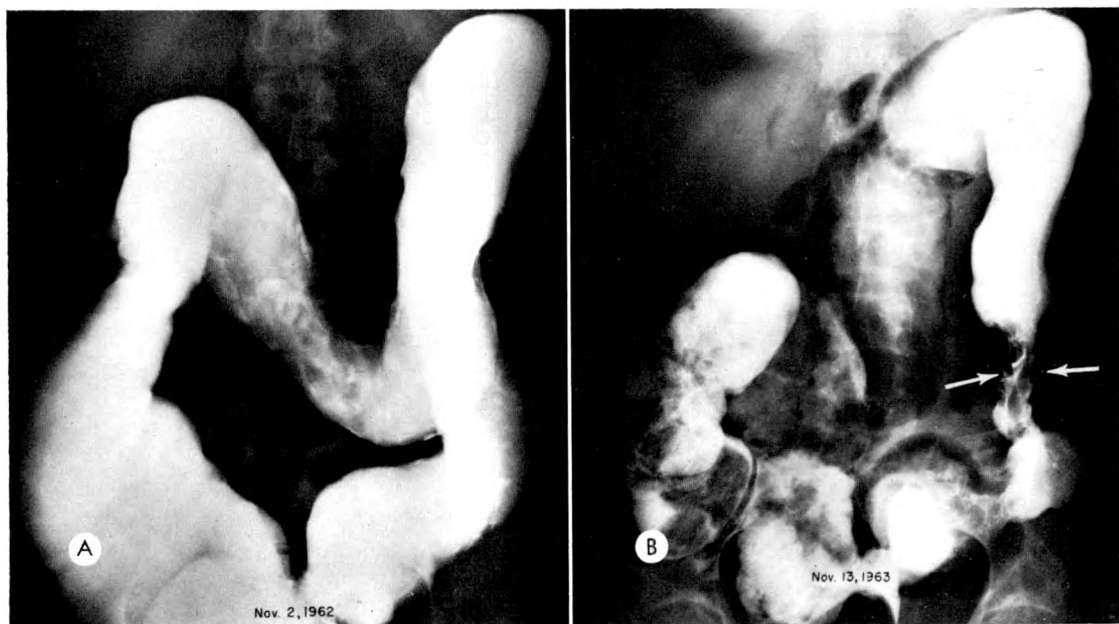


FIG. 16. (*A* and *B*) Diffuse ulcerative colitis. (*A*) Barium enema study showing extensive ulcerative colitis. (*B*) Barium enema study 1 year later showing "carcinoma-like involvement" of the descending colon (arrows).

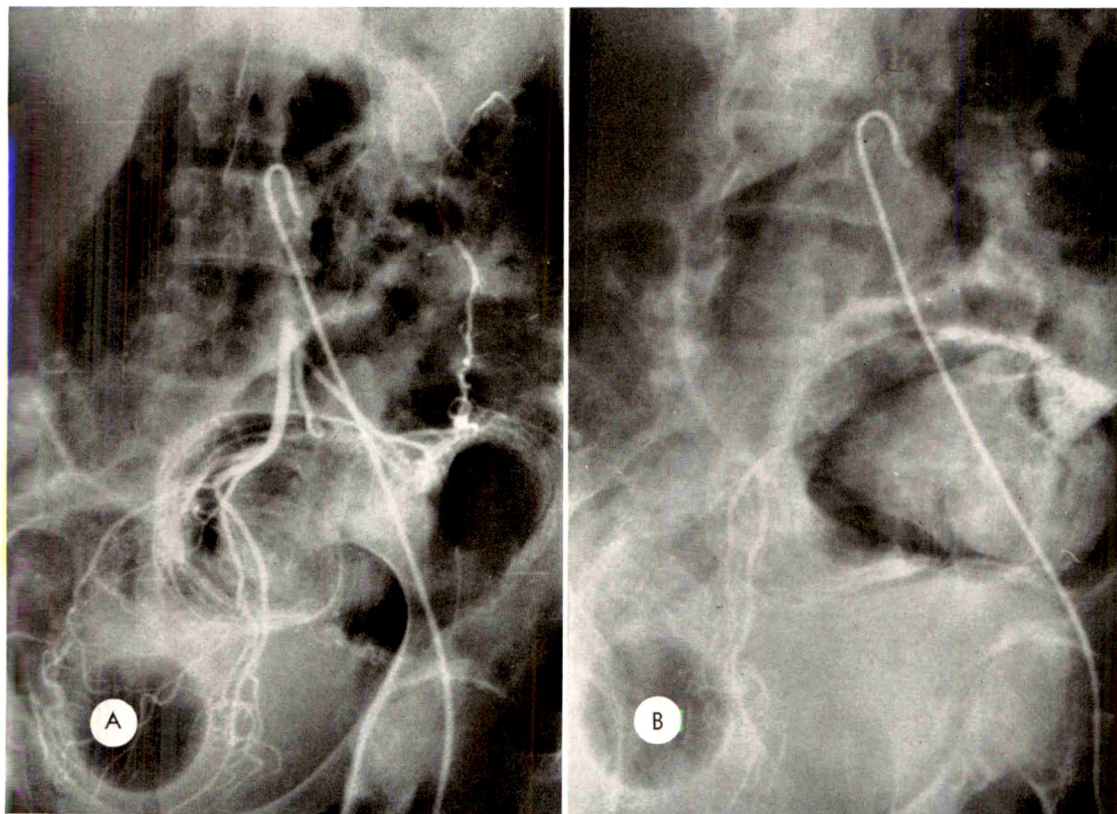


FIG. 17. (A and B) Same case as in Figure 15. Selective inferior mesenteric arteriographic study. Note lack of vessels to the descending colon in the area of the lesion.

enteral solutions. The other enables injection of radiopaque contrast media manually or automatically at variable pressures.

The patient lies comfortably on (9) a motorized remotely controlled rotating cradle which is readily adaptable to any fluoroscopic table. A similar cradle has been developed by Siemens-Reiniger Werke. It permits rotation of the patient from 0 to 90 de-



FIG. 18. Same case as in Figures 15 and 16. Barium enema study prior to surgery. The localized lesion has disappeared. This is an example of pseudotumor, probable reversible vascular occlusion.

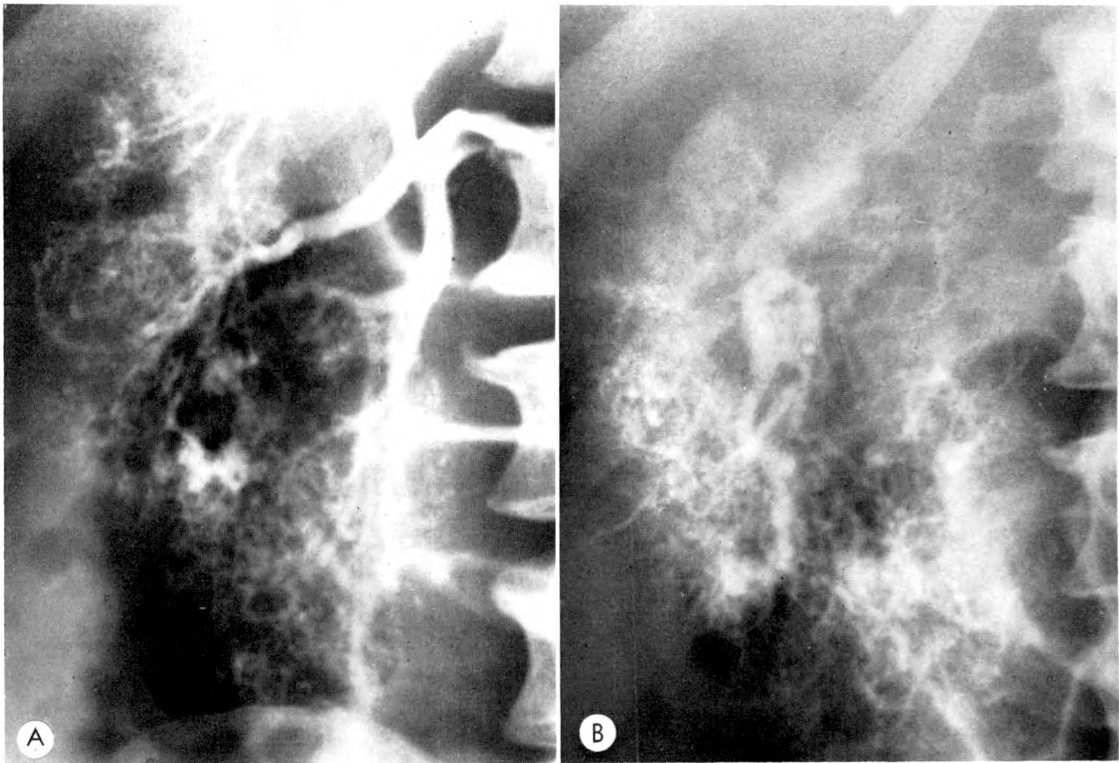
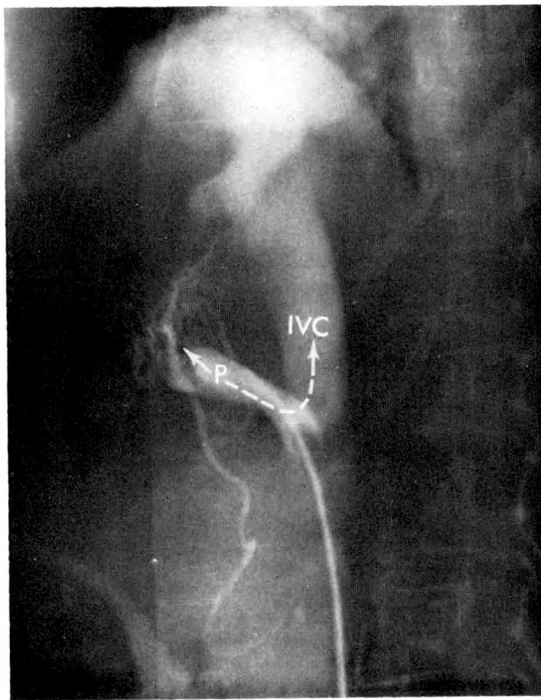


FIG. 19. (A and B) Selective right renal arteriograms. Diffuse renal carcinoma showing characteristic tumor vessels.



grees to either side at variable speeds any time during the procedure. With this mobile cradle, the fluoroscopic positioning of the catheter is facilitated and filming can be done during rotation of the patient.

Figures 1 through 22 illustrate the described technique and applications.



FIG. 20. Direct portography. Examination done following side-to-side portocaval shunt. Injection performed in the portal vein (straight arrow). Note predominant retrograde flow into the inferior vena cava (broken arrow). P=portal vein; IVC=inferior vena cava.

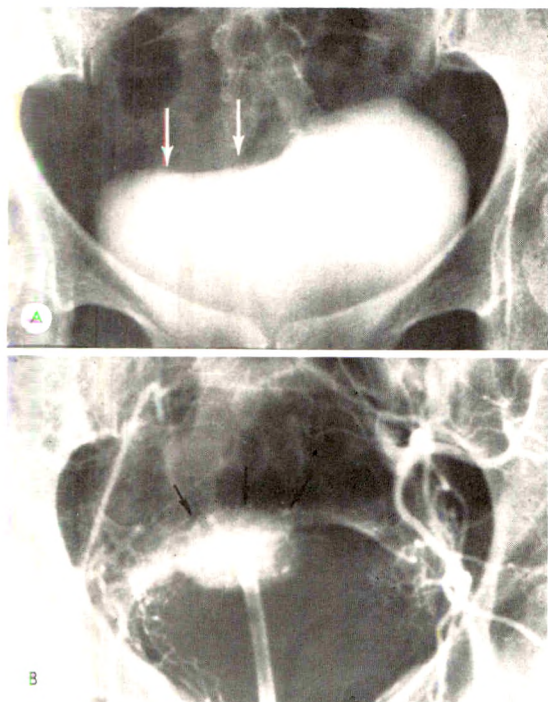


FIG. 21. Bladder carcinoma. (A) Cystogram suggesting "extrinsic defect" in dome of bladder. (B) Bilateral hypogastric arteriographic study with carbon dioxide insufflation of the bladder showing an "iceberg" tumor with invasion of the bladder wall (arrows).

SUMMARY

A technique called "guided angiography" is reported. It has been found to be of use in morphologic and hemodynamic studies: cardiac and vascular pathology, congenital and acquired; mass lesions, tumors versus inflammation and in the differentiation between benign and malignant tumors. The selective introduction of drugs is also facilitated by this method.

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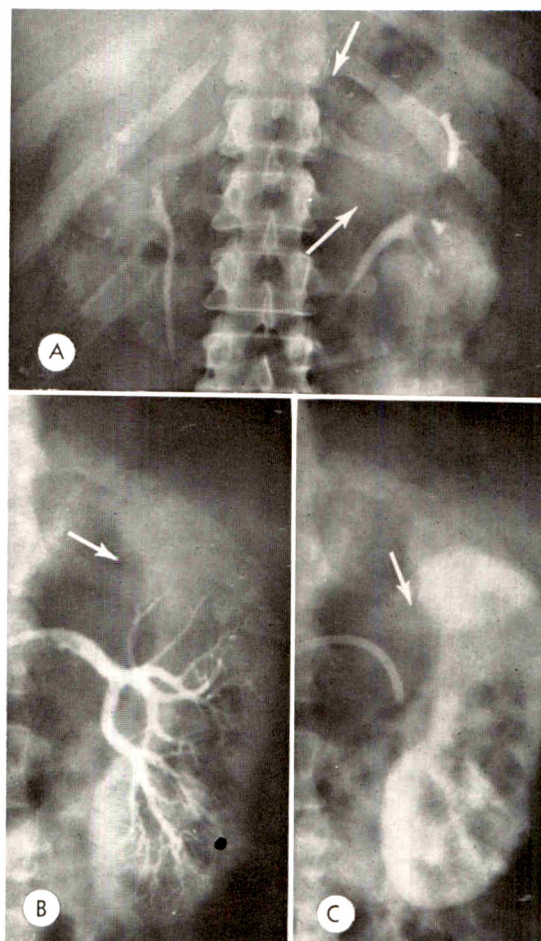


FIG. 22. (A-C) Left renal cyst. Note avascular left upper pole with a negative nephrogram in that area (arrows).

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THE VALUE OF CATHETER ARTERIOGRAPHY IN EVALUATING ARTERIAL INSUFFICIENCY

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WHEELING, WEST VIRGINIA

WHEN a safer method is available, it seems inconsistent with good judgment to subject an already diseased artery to the trauma of direct needle puncture for arteriographic study. Direct needle puncture into the diseased area is usually unnecessary and is potentially hazardous in many cases. Whereas healthy, pulsating arteries may be punctured with ease, a stenotic artery with a diminutive pulse often requires the added insult of multiple punctures.

Although not without hazard, the success and relative safety of catheter aortoarteriography by the Seldinger method are becoming well established.^{1,3,4} This enables the surgeon or radiologist to approach the area of disease via catheter inserted at a more distant point into a relatively normal artery. By following these principles, we have performed over 400 catheter arteriographic studies in the past 20 months with no serious permanent sequelae (Table I).

The following technical approach has been found quite satisfactory:

1. In general, arterial disease in the vicinity of the aortic arch, brachiocephalic, and cranial vessels is best evaluated selectively by a femoral catheter approach. Aorto-iliac and femoro-popliteal lesions are best evaluated using an axillary artery catheterization, preferably the left.

2. Each patient is evaluated prior to the arteriographic procedure and the best approach selected, based on the probable pathology and the location of clinically normal arteries for catheter entrance.

3. When arterial catheterization is performed, preliminary retrograde pressure arterial study is extremely valuable as a "roadmap" prior to catheter insertion, if

TABLE I

DISTRIBUTION OF CATHETER ARTERIOGRAPHY
AND AORTOGRAPHY PROCEDURES
JANUARY, 1963 TO SEPTEMBER, 1964

<i>Carotid</i>	
Femoral catheterization	60
Axillary catheterization	10
<i>Renal</i>	
Femoral catheterization	56
Axillary catheterization	15
<i>Femoro-popliteal</i>	
Axillary catheterization	27
<i>Other Selective Abdominal Arteriographies</i>	
Femoral catheterization	7
Axillary catheterization	3
<i>Aorta (thoracic)</i>	
Femoral catheterization	85
Axillary catheterization	25
<i>Aorta (abdominal)</i>	
Femoral catheterization	83
Axillary catheterization	32
Total	403

the guide wire tip does not easily pass into the aorta (Fig. 1, *A*, *B* and *C*).^{5,6}

4. Preliminary pressure aortography then establishes a further guide for selective arterial catheter insertion (Fig. 2, *A* and *B*; and Fig. 3, *A* and *B*).

This approach has special merit in the study of occlusive disease of the aorto-iliac and femoro-popliteal areas. The procedure is performed as follows:

- A. A normally pulsating axillary artery is cannulated, preferably the left (Fig. 4, *A* and *B*; and Fig. 5).

- B. If the guide wire tip will not easily pass into the aorta, the cannula is advanced slightly, free arterial blood flow ascertained and a retrograde axillary arteriogram is made as a guide to catheter passage (Fig.

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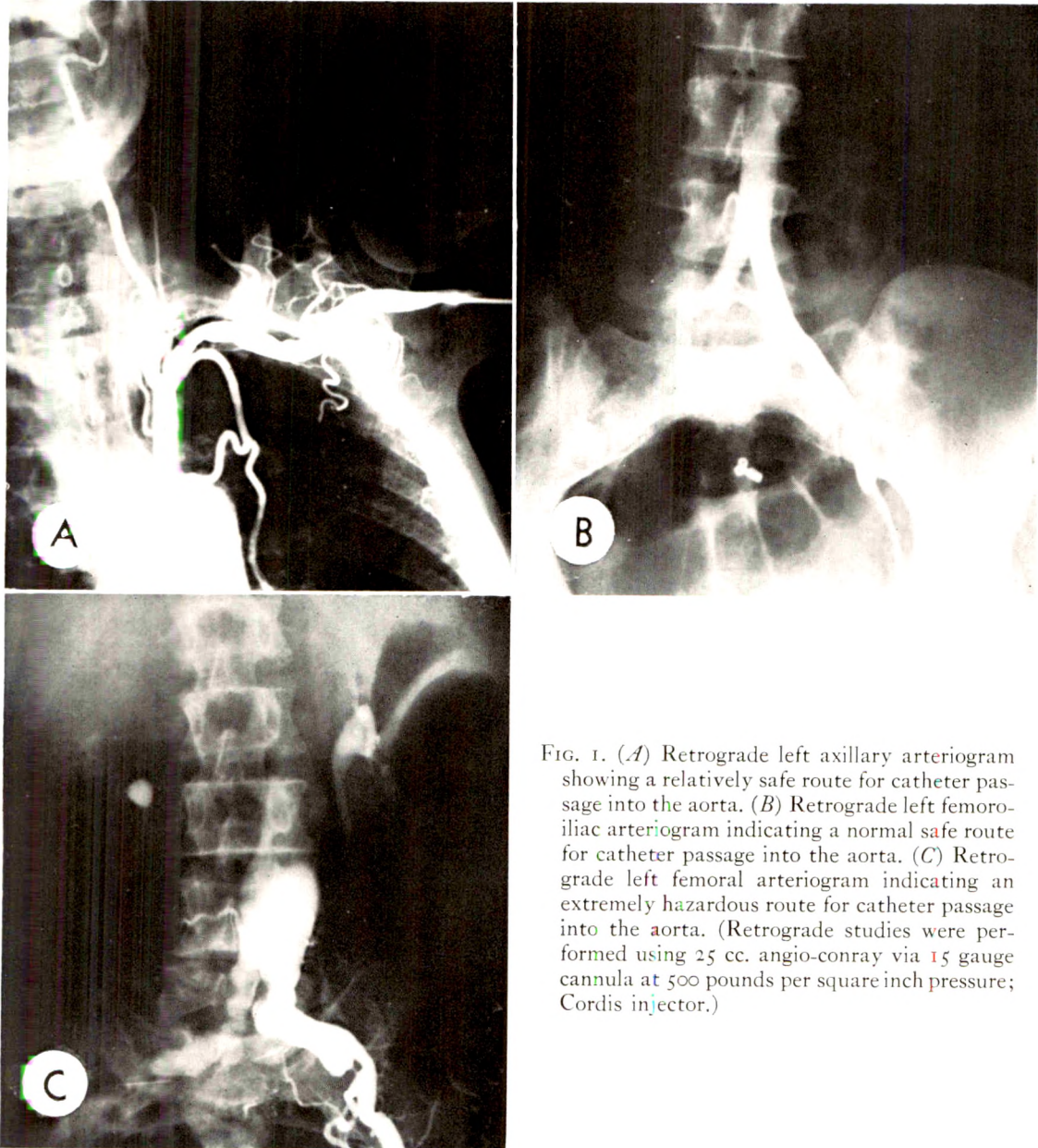


FIG. 1. (A) Retrograde left axillary arteriogram showing a relatively safe route for catheter passage into the aorta. (B) Retrograde left femoroiliac arteriogram indicating a normal safe route for catheter passage into the aorta. (C) Retrograde left femoral arteriogram indicating an extremely hazardous route for catheter passage into the aorta. (Retrograde studies were performed using 25 cc. angio-conray via 15 gauge cannula at 500 pounds per square inch pressure; Cordis injector.)

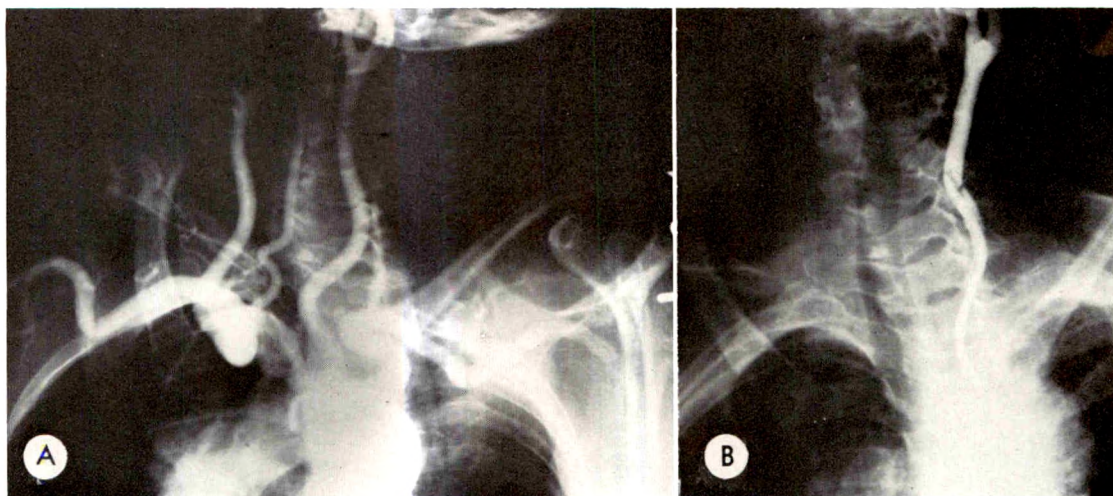


FIG. 2. Fifty-nine year old female referred for left carotid catheter arteriography. (A) Arch aortogram made via left femoral artery catheterization. The tortuosity and kinking of these subclavian arteries would indicate a hazardous route for aortic catheterization via axillary puncture. (B) Catheter selectively placed in left common carotid artery for intracranial arteriography.

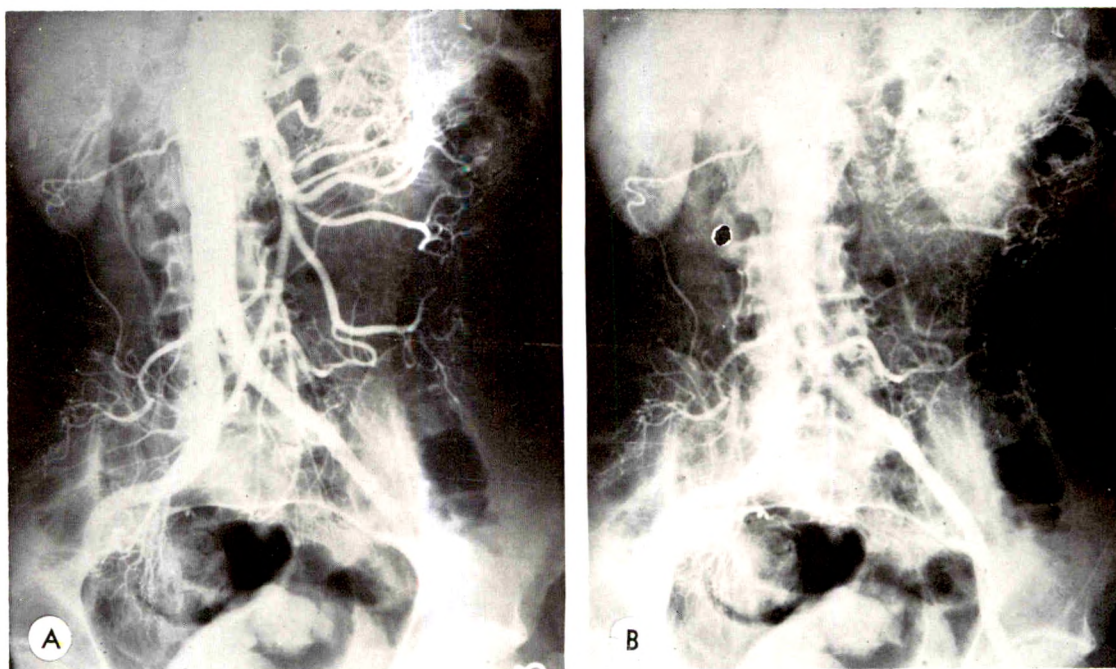


FIG. 3. Aorto-ilio-femoral arteriography in a 52 year old patient performed via catheter inserted through the left axillary artery and advanced into the upper abdominal aorta. Patent distal aorta and ilio-femoral arteries permitted selective catheter placement in the left and right femoral arteries (shown in Fig. 6, A and B). (A) One second after injection of 30 cc. angio-conray at 500 pounds per square inch. (B) One second later.

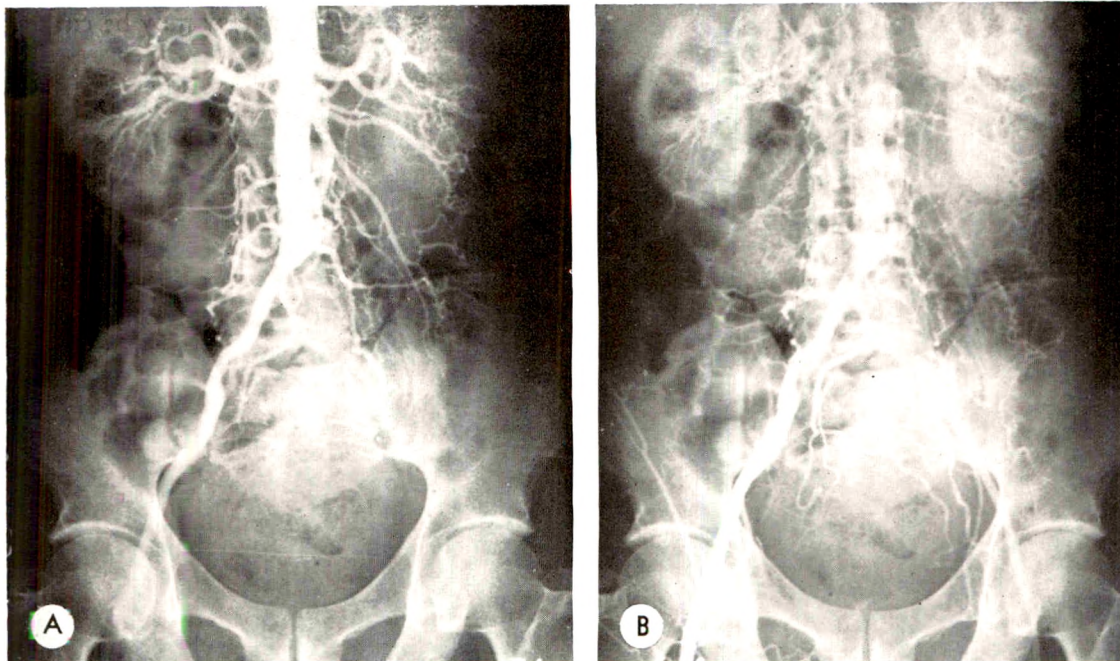


FIG. 4. Aorto-ilio-femoral arteriogram via left axillary catheter in a 58 year old male showing total occlusive lesion of proximal left common iliac artery. (A) One-half second after rapid pressure injection of angioconray. (B) One-half second later.

1A). If a hazardous route is shown, the opposite axillary artery may be substituted, although catheter passage down the aorta from the right side is frequently more difficult, particularly if the aortic arch is tortuous.

C. The catheter tip is gently passed into the upper abdominal aorta. At this point, an antegrade aortogram is made with visualization of the abdominal aorta as well as the iliac and common femoral arteries (Fig. 3, A and B).^{5,6}

D. The safety and wisdom of passing the curved catheter tip into one or both ilio-femoral segments for selective femoro-popliteal arteriography are then easily esti-

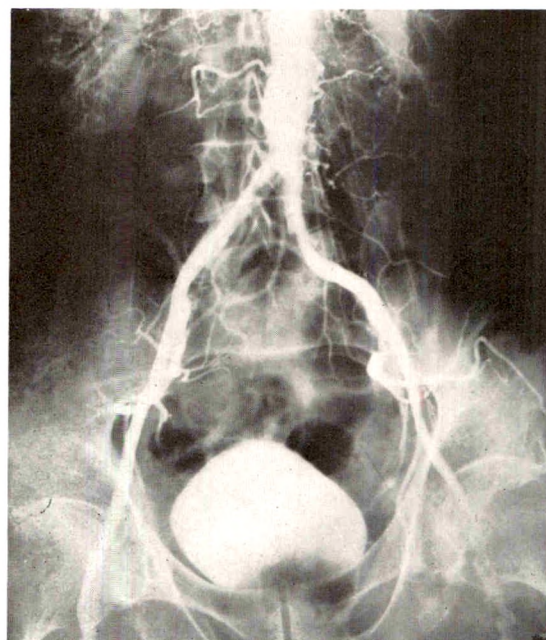


FIG. 5. Sixty-eight year old male. Left axillary catheter aorto-ilio-femoral arteriogram showing extensive occlusive disease of both femoral arteries. Following this, selective catheter injection of both iliac arteries was carefully made for evaluation of the major arterial tree of each thigh and knee region.

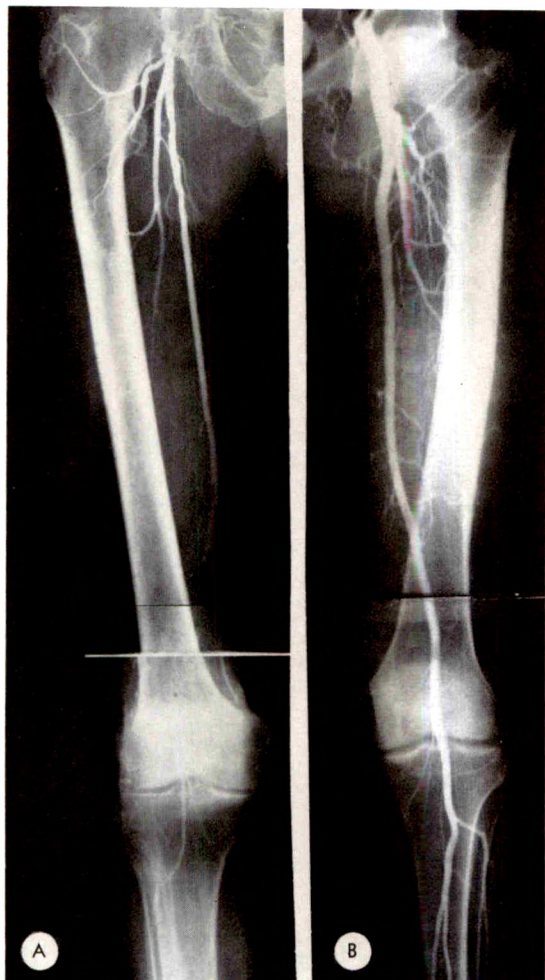


FIG. 6. (A and B) Catheter femoral arteriograms of both lower extremities in 52 year old male whose aorto-iliac segments are shown in Figure 3, A and B. Note the normality of the left arteriogram with femoro-popliteal occlusive disease on the right.

mated. At this point, we use a radioactive circulation timing technique which produces consistently good results in lower extremity arteriography (Fig. 6, A and B).²

E. Although less satisfactory, a clini-

cally normal femoral artery may be catheterized to evaluate the opposite aorto-ilio-femoral area. In such cases, preliminary pressure retrograde femoral arteriograms offer a safe guide (Fig. 1, B and C).

SUMMARY

1. This communication emphasizes an arteriographic technique to avoid direct needle puncture of diseased arteries.
2. Arteriography of abnormal arteries may be safely accomplished by using a more distant location for catheter insertion, if a clinically normal vessel is available.
3. By following this approach, no serious or permanent complications have occurred in 403 catheter procedures.

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ANGIOCARDIOGRAPHIC DIAGNOSIS OF BOTH GREAT VESSELS ORIGINATING FROM RIGHT VENTRICLE*

REPORT OF THIRTEEN ACYANOTIC PATIENTS

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ANGIOCARDIOGRAPHY has become generally recognized as the definitive method for establishing the diagnosis of origin of both great vessels from the right ventricle during life. The technique was first recommended for diagnosis by Witham¹¹ in 1957. It has been extensively used in this center for diagnosis since 1959.^{2,3,4,10} Recent publications have described its value in isolated cases.^{1,5} The purpose of this report is to review and emphasize the roentgen features of ventricular septal defect associated with origin of both great vessels from the right ventricle, to record our experience in 13 acyanotic patients and, thereby, to evaluate the role of contrast roentgenography in the diagnosis of the congenital anomaly.

METHODS AND MATERIALS

Angiocardiography, intravenous and selective (right and left ventricular), was performed in 13 acyanotic patients, 7 females and 6 males. All had complete clinical and laboratory evaluation which included electrocardiographic,⁴ phonocardiographic, roentgenoscopic, and conventional roentgenographic studies in the frontal, lateral, and 2 oblique positions. Cardiac catheterization was performed in 11 of the cases.⁴

RESULTS

Table 1 records the conventional roentgenographic findings. Enlargement of the heart (usually all 4 chambers), of the pulmonary artery, and plethoric lungs were present in 8 of the 13 patients. Three

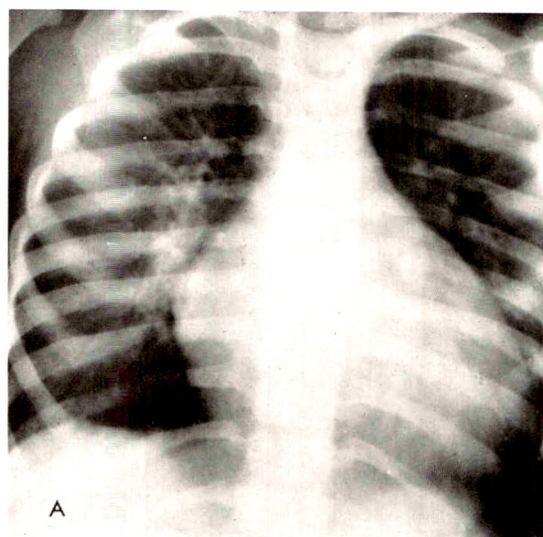


FIG. 1. Case 1. (A) Conventional roentgenogram of the chest showing enlargement of the heart and plethoric lungs.

patients (Case 2, 5, and 12) had normal-sized hearts, but the pulmonary artery was enlarged and the lungs were plethoric in 2 of these, although not in the third (Case 12). Of 2 patients with slight cardiac enlargement (Case 3 and 4), the lung vasculature was plethoric in the former and only slightly increased in the latter. The pulmonary artery appeared normal in position in all patients. No abnormality of the aorta was noted, even in retrospect.

Intravenous angiocardiography (in 2 instances in simultaneous, biplane, frontal and lateral views) was performed in 6 patients (Case 1, 2, 5, 6, 9, and 11). One patient had countercurrent aortography after intravenous angiocardiography to

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TABLE I
ROENTGENOGRAPHIC FINDINGS IN 13 CASES OF ORIGIN OF BOTH GREAT VESSELS FROM RIGHT VENTRICLE

Case No.	Age (yr.)	Sex	Conventional Roentgenography	Angiocardiography	Remarks, Follow-Up, Operation, Autopsy
1	2	F	Frontal study revealed enlarged heart with plethoric lungs (Fig. 1A). Oblique views showed enlargement of ventricles and pulmonary artery	Intravenous angiograms showed enlargement of the right atrium, ventricle, pulmonary artery, and branches (Fig. 1, B and C). Left atrium and ventricle were also enlarged with reopacification of the right heart structures (Fig. 1, D and E). The aorta became opacified from the left ventricle; the aortic valves arose in the right ventricle (Fig. 1, E and F)	Ventricular septum closed without realizing that aorta also arose from the right ventricle. At autopsy, 2 cm. ventricular septal defect of membranous portion was seen. Both atria and ventricles were dilated and hypertrophied; aorta originated from right ventricle above septal defect ^{1,10}
2	9	F	Normal-sized heart in frontal view with prominent pulmonary artery and plethoric lungs. Calcified mediastinal mass in posterior mediastinum (Fig. 2, A and B). Left ventricle enlarged in left anterior oblique view (Fig. 2B)	Intravenous angiograms showed right atrium, ventricle, and pulmonary artery enlargement (Fig. 2, C and D). Left ventricle also enlarged with left-to-right shunt at ventricular level with aorta arising from right ventricle (Fig. 2, E and F)	Neuroblastoma removed on April 22, 1959. On May 9, 1961, septal defect corrected with open heart surgery and a special sling prosthesis to carry left ventricular blood to aorta was inserted. Patient well and asymptomatic—3 years ¹¹
3	17	F	Slightly enlarged heart with greatly enlarged pulmonary artery and branches, and plethoric lungs	Selective biplane left ventricular angiograms showed opacification of aorta from the left ventricle via a tunnel which produced a distorted appearance of outflow tract of left ventricle. ¹ Intravenous angiogram revealed normal pulmonary artery arising from right ventricle	Eleven year follow-up; now 21 years old. Patient asymptomatic and recently had interruption of pregnancy. Cardiac surgery not advised because of age. ¹
4	1	F	Heart and pulmonary artery slightly enlarged with slight increase in pulmonary vasculature	Intravenous angiograms showed enlargement of right atrium, ventricle, and pulmonary artery. Selective right ventricular angiograms disclosed both great vessels from right ventricle, left-to-right shunt at ventricular level, and deformed outflow tract due to left ventricular aortico-root tunnel	Now age 2½ years, still acyanotic, and doing well; operation deferred ¹
5	14	F	Normal-sized heart with enlarged pulsating pulmonary artery without engorgement of pulmonary vessels	Intravenous angiograms showed enlargement of both sides of the heart, left-to-right shunt at ventricular level, and aorta arising from the right ventricle (Fig. 3, A, B and C)	Now a college student, aged 20 years, with minimal cyanosis at rest. Operation probably contraindicated
6	2	M	Enlarged heart especially left ventricle (Fig. 4, A, B and C). Esophagus displaced by an enlarged left atrium (Fig. 4, A and C). Lung vasculature prominent	Intravenous angiograms showed enlarged right atrium, ventricle, and pulmonary artery (Fig. 4, D and E). Aorta faintly opacified from right ventricle (Fig. 4D). Left atrium markedly enlarged (Fig. 4F). Left ventricle enlarged with left-to-right shunt at ventricular level with origin of aorta from right ventricle (Fig. 4G)	Schoolboy, now 5 years old, still asymptomatic and acyanotic. Surgery deferred because of co-existing mitral stenosis

TABLE I (Continued)

Case No.	Age (yr.)	Sex	Conventional Roentgenography	Angiocardiography	Remarks, Follow Up, Operation, Autopsy
7	17	F	Enlarged heart and prominent pulsatile pulmonary artery and branches. Pulmonary vasculature plethoric and all cardiac chambers enlarged	Intravenous and selective right ventricular angiocardiograms showed huge right ventricle and pulmonary artery (Fig. 5A). Aorta not enlarged originating from right ventricle and becoming opacified from left ventricle (Fig. 5, B and C)	Now 20 years old and asymptomatic; declined surgery
8	2	M	All 4 cardiac chambers markedly enlarged. Pulmonary artery pulsatile and prominent, and pulmonary circulation engorged (Fig. 6A)	Intravenous angiocardiograms showed all cardiac chambers enlarged (Fig. 6, B-E). Pulmonary artery twice diameter of aorta. Lungs were plethoric (Fig. 6, B and C). The aorta opacified via a tunnel from left ventricle (Fig. 6, E and F). Countercurrent aortograms excluded patent ductus arteriosus	Now age $2\frac{1}{2}$ years, still acyanotic. Though in heart failure as an infant, he is now doing well; operation deferred
9	3	M	Enlarged cardiac chambers, pulmonary artery, and pulmonary vasculature	Intravenous angiocardiograms showed enlarged right atrium, ventricle, pulmonary artery, and branches (Fig. 7, A and B). Left atrium and ventricle also enlarged with a left-to-right shunt at ventricular level (Fig. 7C). Left anterior oblique view showed aorta originating from the right ventricle (Fig. 7D)	Surgery postponed because of age
10	9	M	Heart massively enlarged, plethoric lungs with enlargement of the cardiac chambers and both great vessels. Both great vessels widely expansile	Huge left-to-right shunt on intravenous angiocardiographic study. Retrograde selective aortography established that aorta originated from right ventricle with aortic insufficiency and marked regurgitation into right ventricle [†]	Two years later severe cardiac failure necessitated operation to correct aortic insufficiency and double outlet right ventricle. Died after surgery; autopsy confirmed angiographic studies [†]
11	2 mo.	F	Heart enlarged, markedly plethoric lungs, and enlargement of all chambers	Simultaneous biplane frontal and lateral intravenous angiocardiograms showed an enormous left-to-right ventricular shunt with normal sequence of opacification of great vessels. Right ventricular origin of aorta was not recognized, probably because an oblique study was not made	Died after 5 months with chronic heart failure and pneumonia. Autopsy proved diagnosis of double outlet right ventricle with ventricular and atrial (secundum) defects
12	10	M	No cardiac enlargement, pulmonary artery enlarged but no plethora of lungs (Fig. 8, A and B)	Selective right ventricular angiocardiograms in frontal and lateral views showed inconclusive data regarding origin of aorta (Fig. 8, C and D). However, the left anterior oblique study was definitive (Fig. 8E). (The selective angiocardiographic studies were performed by Dr. Harry L. Stein.)	Operation within a year appeared successful but patient died suddenly and unexpectedly on fifth postoperative day. At autopsy, position of hammock prosthesis good; extensive pulmonary vascular obliteration present; diagnosis verified
13	4	M	Enlarged heart with markedly engorged lung vessels (Fig. 9, A and B)	Selective left ventriculograms revealed details of double outlet right ventricle (Fig. 9, C and D). Selective retrograde aortogram showed hypoplasia of transverse aortic arch with coarctation of the aorta and patent ductus arteriosus (Fig. 9E). (The selective angiocardiographic studies were performed by Dr. Harry L. Stein.)	First stage operation corrected the coarctation of aorta with a prosthesis; ductus divided. Second stage operation corrected defect with hammock-like prosthesis but patient died of intracranial hemorrhage of unknown cause. Autopsy confirmed diagnosis

evaluate a murmur of aortic insufficiency (Case 8). Two patients had both intravenous and right heart selective angiocardiology (Case 4 and 7). One patient had intravenous angiocardiology and retrograde aortography (Case 10). One patient had both intravenous and selective left heart angiocardiology (Case 3). Selective left ventricular angiocardiology alone was performed in 2 patients. In Case 13 the diagnosis of coexisting co-

arctation of the aorta and patent ductus arteriosus was established by withdrawing the catheter from the ascending to the descending aorta and opacifying of the lesions (Fig. 9E).

In each instance, angiocardiology showed that the pulmonary artery arose normally from the outflow tract of the right ventricle (Fig. 1, *B* and *C*; 2*D*; 4, *D* and *E*; 5*A*; 6, *B* and *C*; and 7, *A* and *B*). Only 2 patients (Case 4 and 6) (Fig. 4*D*)

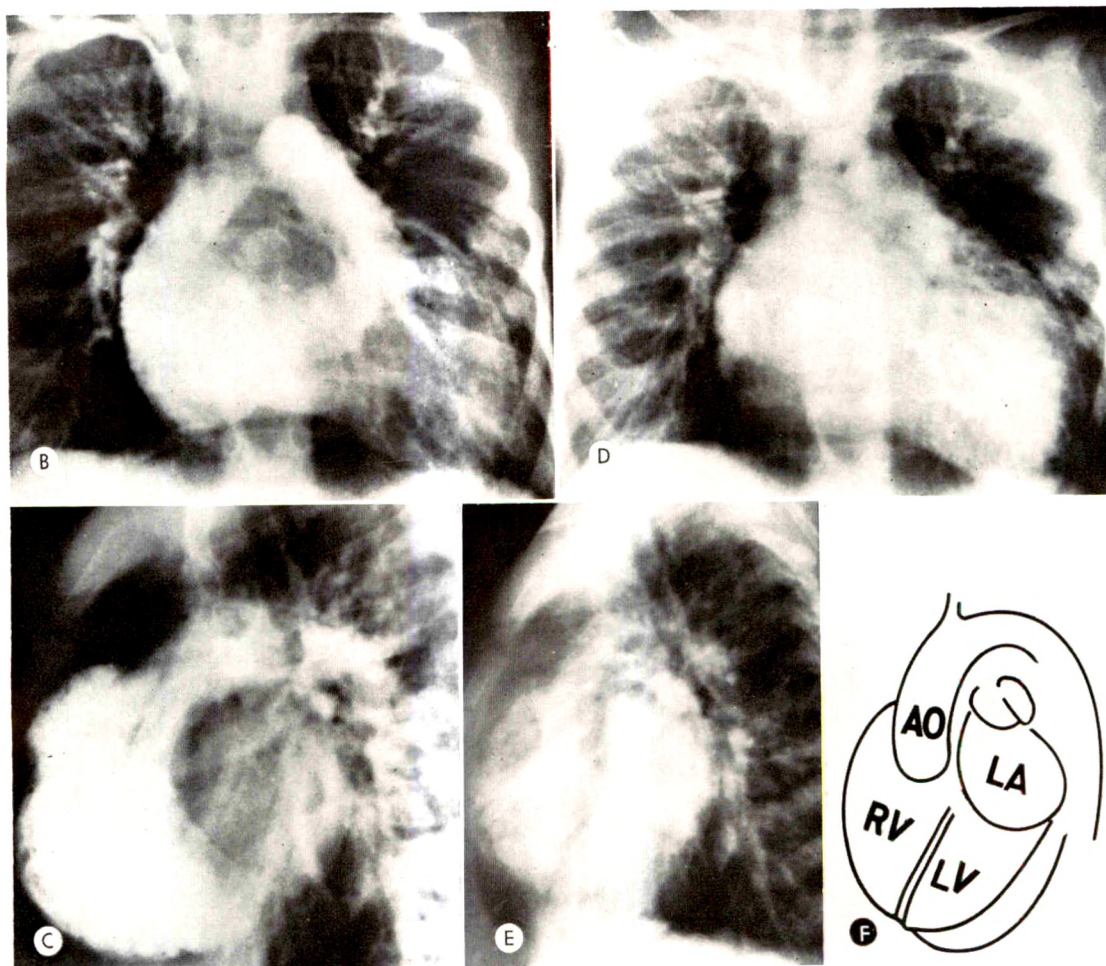


FIG. 1. (*B*) Frontal intravenous angiogram showing enlargement of the right atrium, ventricle, pulmonary artery, and branches. The pulmonary artery arises in a usual fashion from the outflow tract of the right ventricle. (*C*) Left lateral angiogram showing the enlarged right ventricle and pulmonary artery. (*D*) Frontal angiogram showing enlargement of the left atrium and ventricle. The root of the aorta is obscured by the left atrium, but the ascending aorta is in its usual position. There is reopacification of the right ventricle, pulmonary artery, and branches. (*E*) Left anterior oblique angiogram. (*F*) Diagram of *E*, showing the enlarged left atrium (LA), left ventricle (LV) with reopacification of the right ventricle (RV). The aorta (AO) arises from the inflow tract of the right ventricle.

showed early opacification of the aorta. In all other instances the aorta became opacified in regular normal sequence (Fig. 1, *D* and *E*; 2*E*; 3, *A* and *B*; 4, *F* and *G*; 5*B*; 6, *D* and *E*; 7, *C* and *D*; 8, *C*, *D* and *E*; and 9, *A* and *B*). In frontal view, it was often impossible after intravenous angiocardiology to recognize the abnormal position of the root of the aorta in the right ventricle because the left atrium was opacified at the same time. However, following selective injection of contrast medium into the left ventricle, the deformed outflow tract of the left ventricle and the rightward displacement of the aortic valve were well seen.⁴ In the lateral, and especially in the left anterior oblique view, the end-on position of the ventricular septum permitted visualization of the aortic valve and the aorta arising from the right ventricle in an anterior, transposed position in the same plane as, or in front of, the pulmonary artery (Fig. 1*E*; 2*E*; 3*B*; 4*G*; 5*B*; 6*E*; 7*D*; 8, *D* and *E*; and 9, *C* and *D*). Especially in the selective lateral and oblique studies (Fig. 8, *D* and *E*; and 9, *C* and *D*), the streaming of blood from the left ventricle through a septal defect into the aortic valve created a tunnel-like effect, a sign of considerable diagnostic importance.

DISCUSSION

A diagnosis of the origin of both great vessels from the right ventricle associated with a ventricular septal defect is difficult to make on clinical grounds when the patient is acyanotic.^{2,4} Conventional roentgenography may show cardiac enlargement when the shunt is large, and, if the patient is in heart failure, the lungs, too, are frequently plethoric. These findings, however, do not differ greatly from those of a simple ventricular septal defect alone. A significant finding on cardiac catheterization is a systemic pressure in the right ventricle. This makes angiocardiology mandatory, for only with this technique can the origin of both great vessels from the right ventricle be visualized during life.⁴

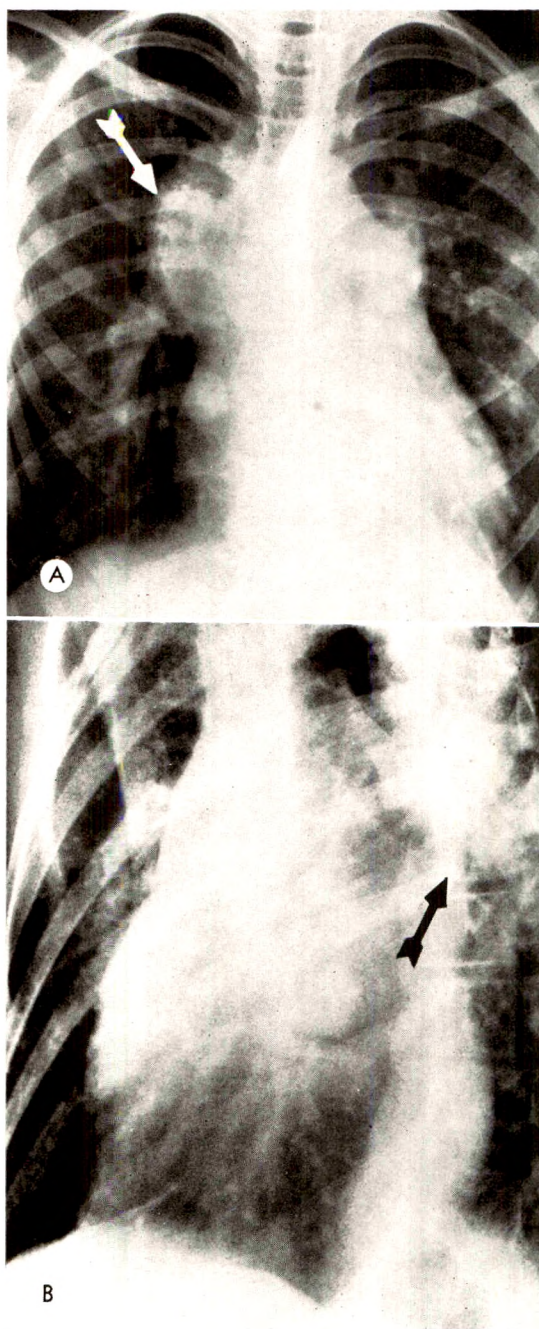


FIG. 2. Case 2. (A) Conventional frontal teleroentgenogram showing enlargement of the heart, pulmonary artery, and pulmonary vasculature. A large calcific mass is in the right superior mediastinum (arrow). (B) Left anterior oblique roentgenogram demonstrating enlargement of the right and left ventricles. The mass (arrow) is in the posterior mediastinum overlying the spine. After excision it was found to be a neuroblastoma.¹⁰

Because of the high degree of mixing of the blood by the left-to-right shunt, the diluted contrast material following intravenous angiocardiology may obscure the tunnel-like streaming of contrast material from the left ventricle across the septal

defect into the right ventricle at the root of the aorta (Fig. 8E; and 9, C and D). Accordingly, selective catheter injection of contrast medium into either the right or left ventricle is recommended. Although the transposed position of the aortic root,

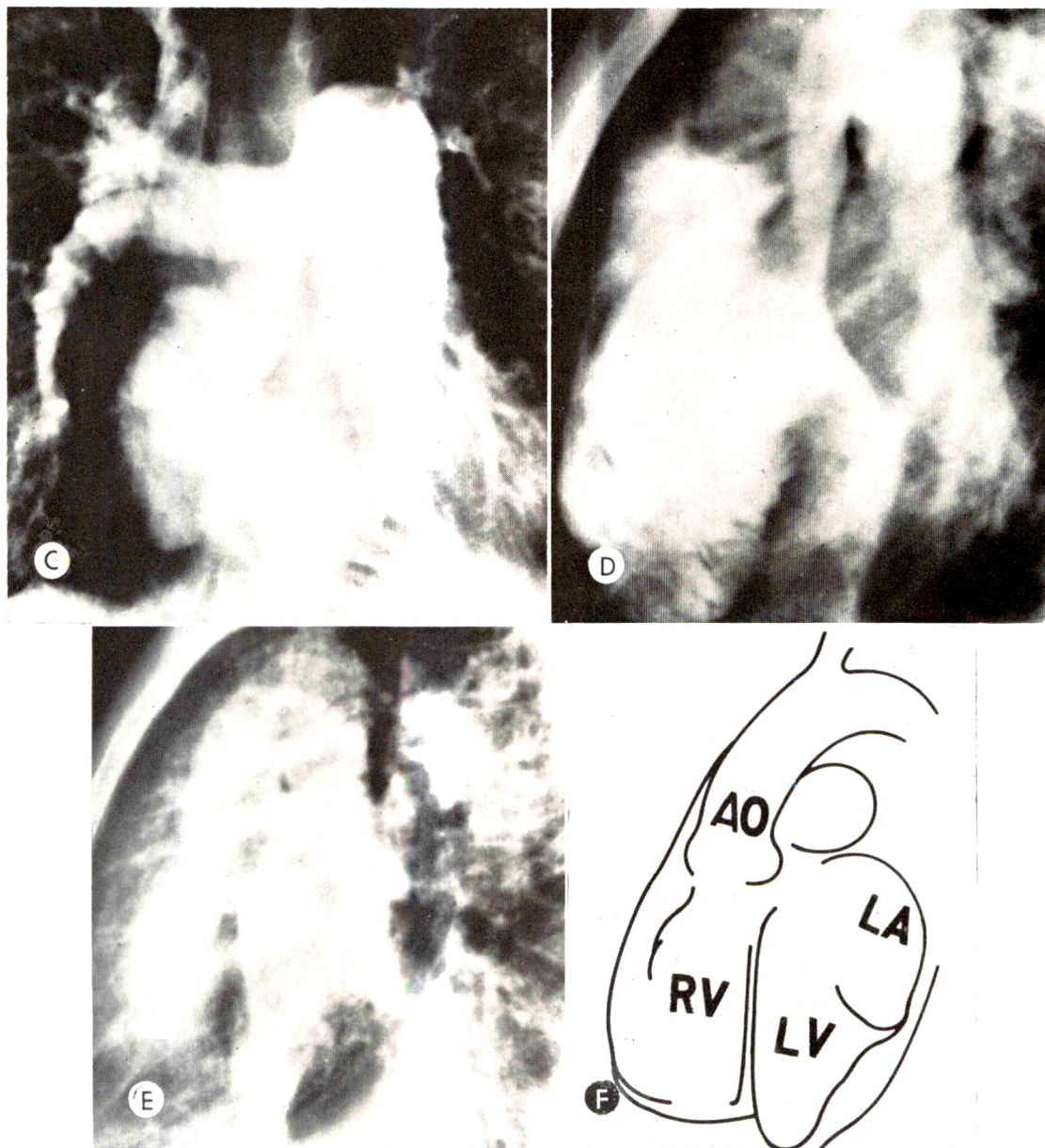


FIG. 2. (C) Frontal intravenous angiogram revealing enlargement of the right atrium, ventricle, pulmonary artery, and branches. (D) The left anterior oblique angiogram also shows enlargement of the right heart chambers and the pulmonary arterial system. (E) The left-to-right shunt at the ventricular level with origin of the aorta from the inflow region of the right ventricle is evident. (F) Diagram of E. (Fig. 2, A and E reproduced with permission of the publisher.¹⁰)

originating to the right of the ventricular septum, may be well demonstrated with the intravenous method (Fig. 2E; 3B; 4G; 5B; 6E; and 7C), this technique cannot achieve the details of the tunnel-like streaming of contrast material produced by selective ventriculography.⁴

Only in Case 11 did angiocardiology fail to show both great vessels arising from the right ventricle. This is probably because there was a secundum type atrial septal defect as well as a ventricular septal defect, and the excessive amount of left-to-right shunting of contrast material made it impossible to establish the diagnosis, even retrospectively. In this case, only a biplane study in frontal and lateral views was made. Failure to secure a left anterior oblique study may have been responsible for the error because the lateral view is not as satisfactory as the left anterior oblique position for demonstrating the plane of the ventricular septum. Although Case 1 showed the classic findings of origin of both great vessels from the right ventricle (Fig. 1E), it was recognized only retrospectively after surgical repair of the ventricular septal defect proved fatal.¹⁰ This occurred before the entity of acyanotic ventricular septal defect with normal position of the pulmonary artery and transposition of the aorta was recognized.² Subsequently, surgical repair of the septal defect and creation of a hammock-like prosthesis to provide for aortic blood flow has been accomplished;^{7,10} a preoperative diagnosis by means of contrast studies was of great importance in identifying the abnormal position of the aorta.

When the amount of pulmonary arterial resistance is high, repair of the septal defect and creation of the special sling prosthesis may not always restore the pulmonary pressure and vessels to a normal state. Figure 8A (Case 12) showed only mild increase in the size of the pulmonary arteries and clear peripheral lung fields, suggesting high pulmonary resistance and small left-to-right shunt. Nevertheless, operation was undertaken because the

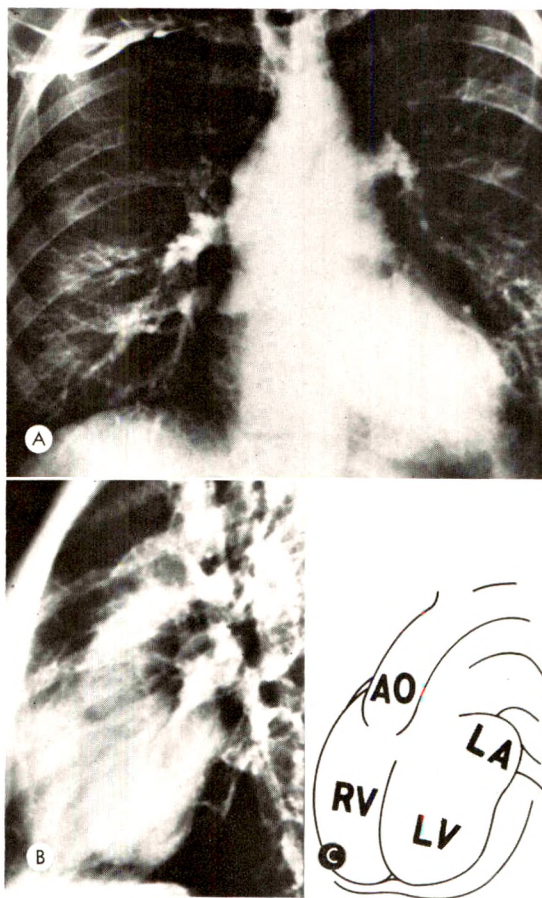


FIG. 3. Case 5. (A) Frontal intravenous angiogram showing an enlarged left ventricle. The origin of the aorta is obscured by the opacified left atrium. (B) In the left anterior oblique angiogram, the origin of the aorta from the right ventricle is evident. (C) Diagram of B.

findings at two cardiac catheterizations a few months apart gave evidence of large pulmonary blood flow. Death occurred unexpectedly on the fourth postoperative day. Autopsy disclosed severe and diffuse pulmonary vascular obstructive disease. Such a situation was also encountered by Lucas *et al.*⁶ Operation appeared contraindicated in 2 patients (Case 3 and 7), who were over 20 years of age, because of the elevation of pulmonary vascular resistance.

Neufeld and co-workers^{8,9} have emphasized that mitral valve deformities may occur with ventricular septal defects that are associated with right ventricular origin

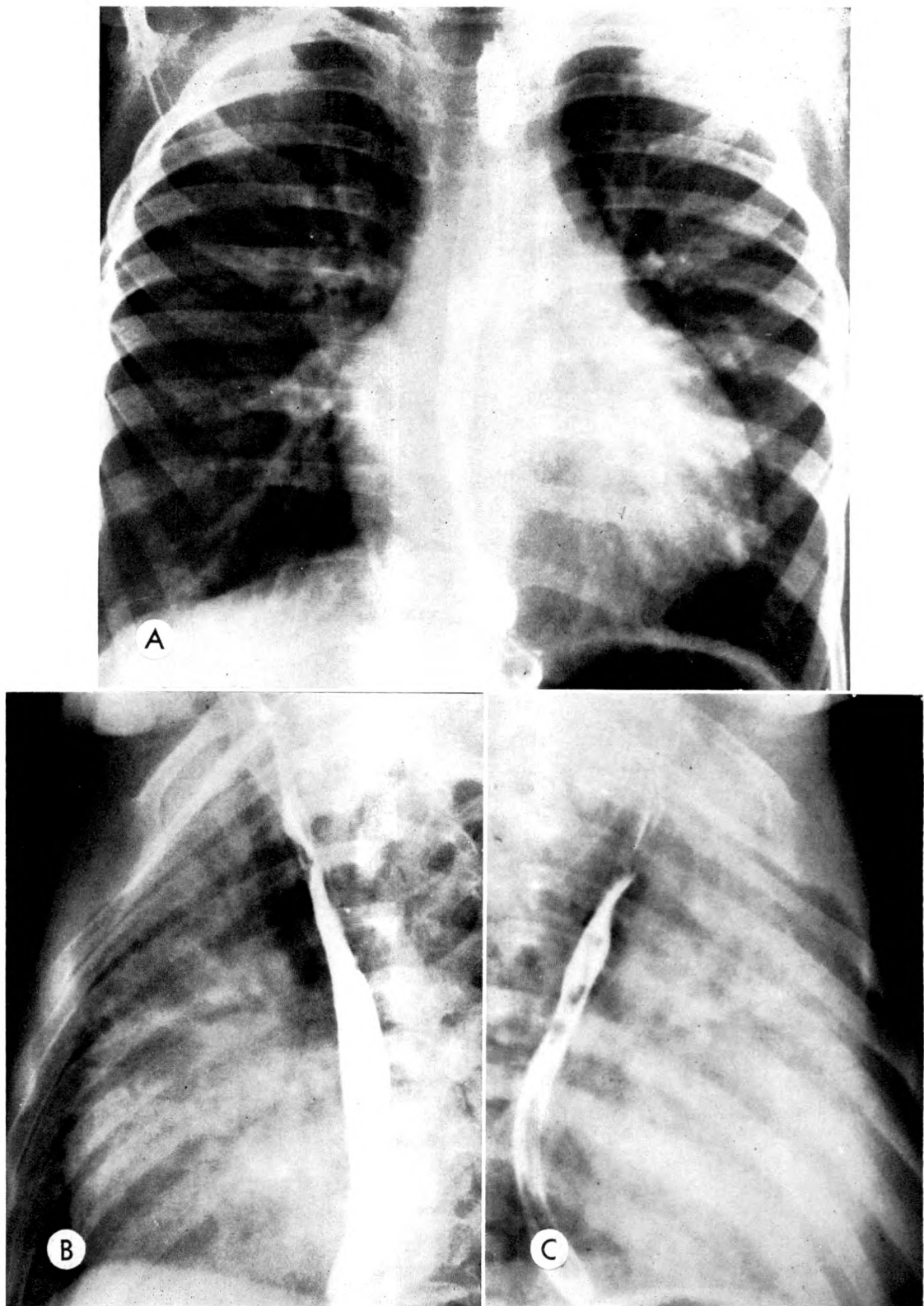


FIG. 4. Case 6. (A) Frontal esophagram showing cardiomegaly, enlargement of the pulmonary artery, and plethora of the lungs. (B) The left anterior oblique roentgenogram with esophagram also shows right and left ventricular enlargement. (C) The right anterior oblique view confirms the right ventricular enlargement. The esophagram demonstrates displacement of the barium column by a large left atrium.

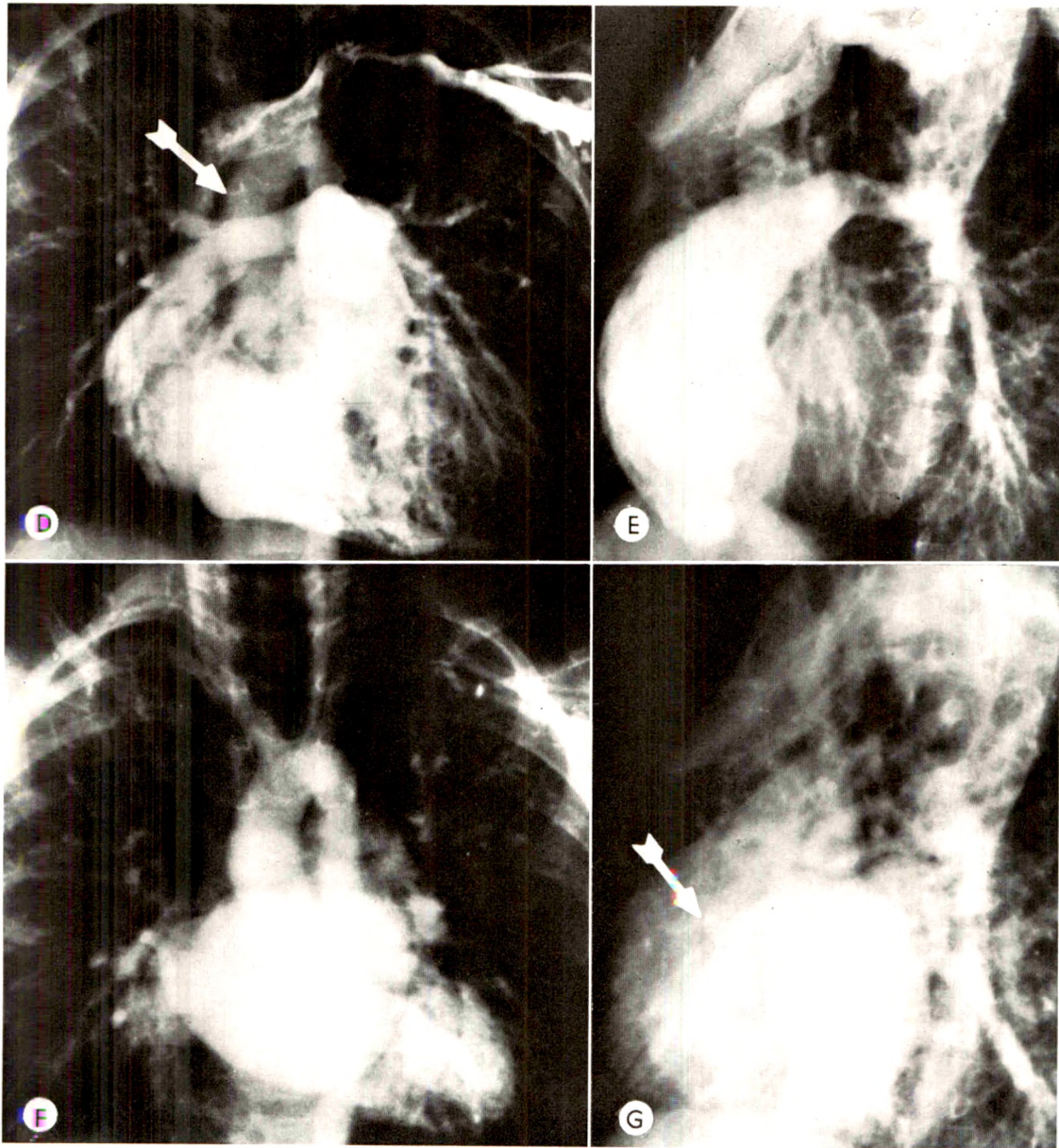


FIG. 4. (D) Frontal intravenous angiogram revealing a large right atrium, ventricle, pulmonary artery, and branches. Note that the aorta (arrow) is prematurely opacified. (E) Left anterior oblique angiogram also showing the large right ventricle and pulmonary artery. In this study, the aorta was not opacified early. (F) Frontal angiogram revealing the huge left atrium due to mitral stenosis. (G) Left anterior oblique angiogram also showing the enlarged left atrium. The aortic cusps (arrow) arise from the inflow tract of the right ventricle.

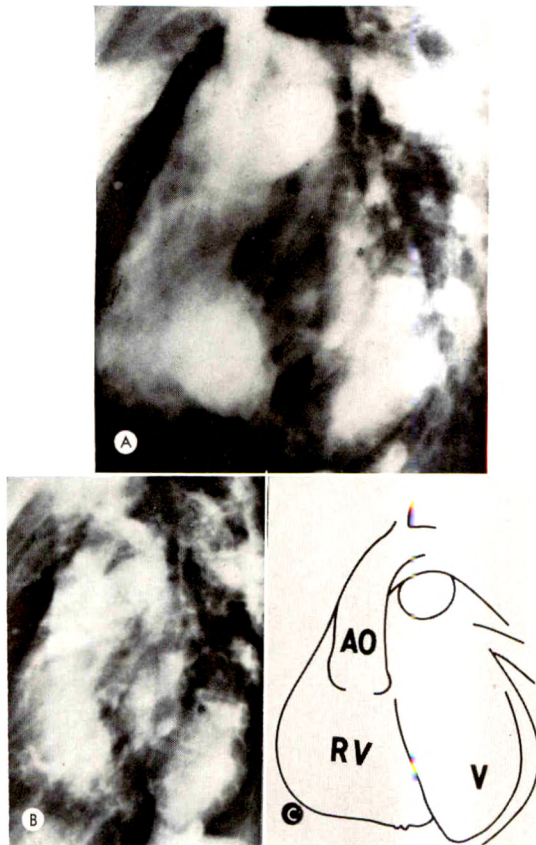


FIG. 5. Case 7. (A) Left anterior oblique intravenous angiogram showing a huge right ventricle and pulmonary artery. (B) When the left ventricle was filled, the aorta, which arose from the right ventricle, became opacified via the septal defect. (C) Diagram of B.

of both great vessels. The coexistence of mitral stenosis in Case 6 is noteworthy. Because the patient is still asymptomatic and acyanotic, operation, which should also include repair of the mitral stenosis, has been postponed.

Case 10, in addition to the great vessel anomalies, was complicated by severe aortic regurgitation probably due to a perforated cusp from previous bacterial endocarditis. Operation, performed as a last resort, was unsuccessful because of the long period of time required to repair both lesions during open heart surgery.⁴ The last patient in the series had a complicating infantile type of coarctation and a patent ductus arteriosus. A first stage operation

for repair of the coarctation and division of the ductus was successful (Table 1).

Angiocardiography is also capable of differentiating other types of transposition of the great vessels from that discussed above. In the Taussig-Bing anomaly, there is transposition of the aorta while the pulmonary artery arises normally from the outflow tract of the right ventricle or straddles the anterior ventricular septal defect. In this situation, both vessels opacify from the right ventricle, the aorta more densely than the pulmonary artery, where contrast medium is diluted by the stream of unopacified blood entering it through the left ventricle and septal defect. The diagnosis of complete transposition of the great vessels need not be a problem for usually the origin of only the aorta from the right ventricle is distinct. The origin of the pulmonary artery from the left ventricle can be recognized if sufficient shunting into the left side of the heart occurs during angiocardiography. The severe cyanosis in complete transposition of the great vessels is also striking and is in contrast to the lack of visible cyanosis in the situation described above. Patients with corrected transposition may be acyanotic; their angio-

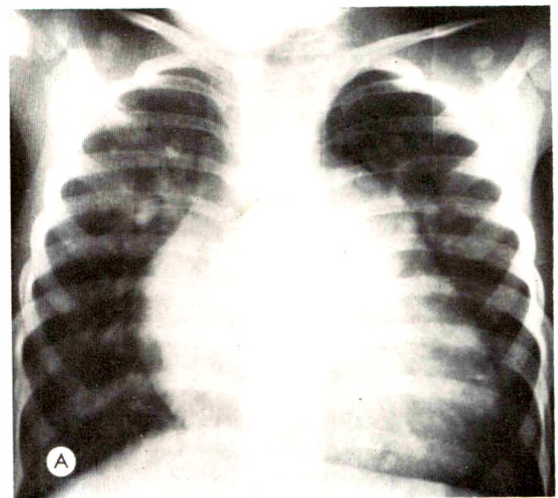


FIG. 6. Case 8. (A) Frontal conventional teleroentgenogram showing generalized enlargement of the heart, pulmonary artery, and pulmonary vasculature.

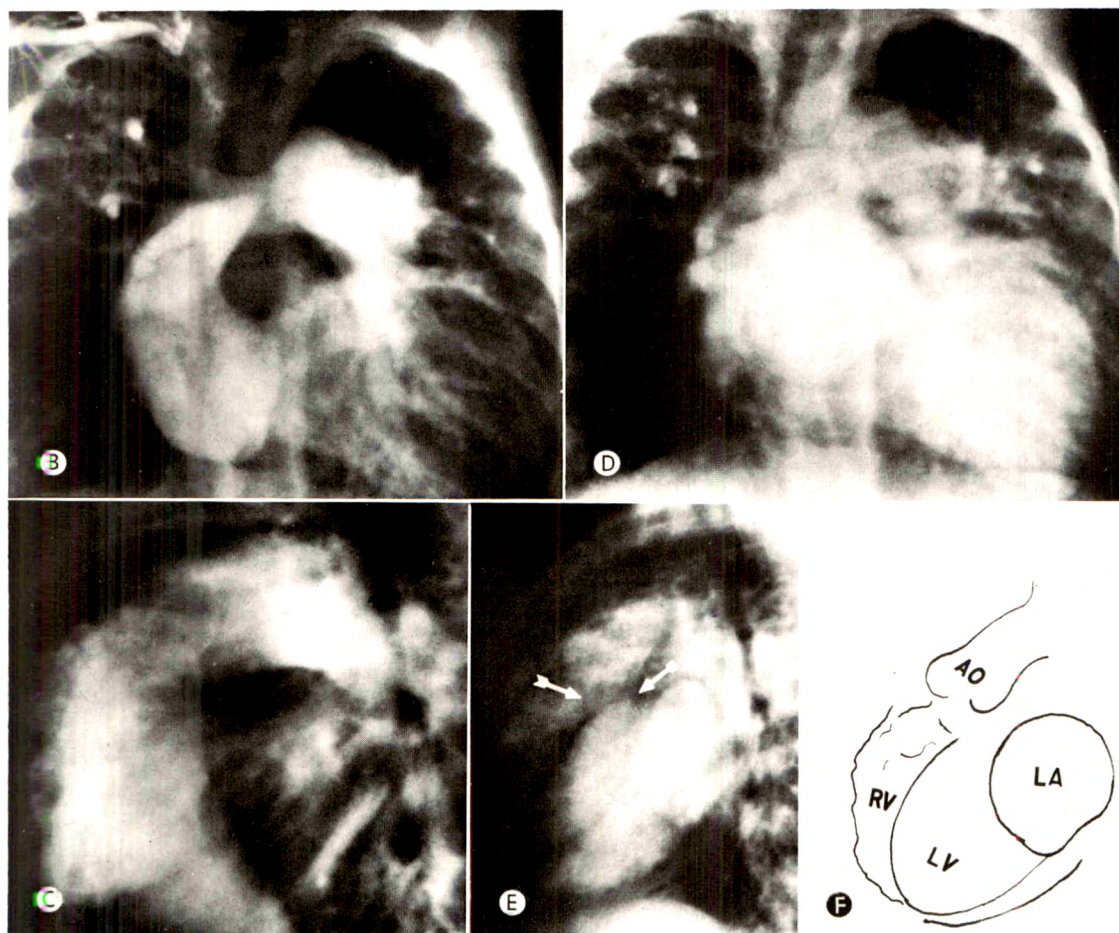


FIG. 6. (B) Frontal intravenous angiocardiogram showing an enlarged right atrium, ventricle, pulmonary artery, and branches. (C) Left anterior oblique angiocardiogram also showing the right ventricular and pulmonary arterial enlargement. (D) Angiocardiogram demonstrating left atrial and ventricular enlargement. There is reopacification of the right ventricle and pulmonary arterial system. The ascending aorta appears to be in its normal position. (E) Left anterior oblique angiocardiogram, however, shows that the origin of the aorta is from the right ventricle. Arrows indicate the tunneling of contrast medium through the septal defect into the aorta. (F) Diagram of E.

cardiograms, however, are revealing for usually the contours of the inverted ventricles and the position of the great vessel with an anterolateral aorta and posteromedial pulmonary artery are distinctive.

Patients who have both great vessels arising from the right ventricle and have pulmonic stenosis may mimic tetralogy of Fallot. Angiocardiography is capable of establishing the diagnosis preoperatively.⁶ Selective angiocardiography, however, especially in the left anterior oblique view, is desirable. The clear-cut origin of the entire

aortic valve from the right ventricle differentiates the anomaly from the overriding of the ventricular septum by the aorta in the tetralogy of Fallot. In 1 of 2 cyanotic patients with pulmonic stenosis and with origin of the great vessels from the right ventricle, the preoperative diagnosis was missed probably because only frontal and lateral intravenous angiocardiograms were made. In the second case, both intravenous and selective right ventricular studies in frontal and lateral views did not permit the diagnosis even after a retro-

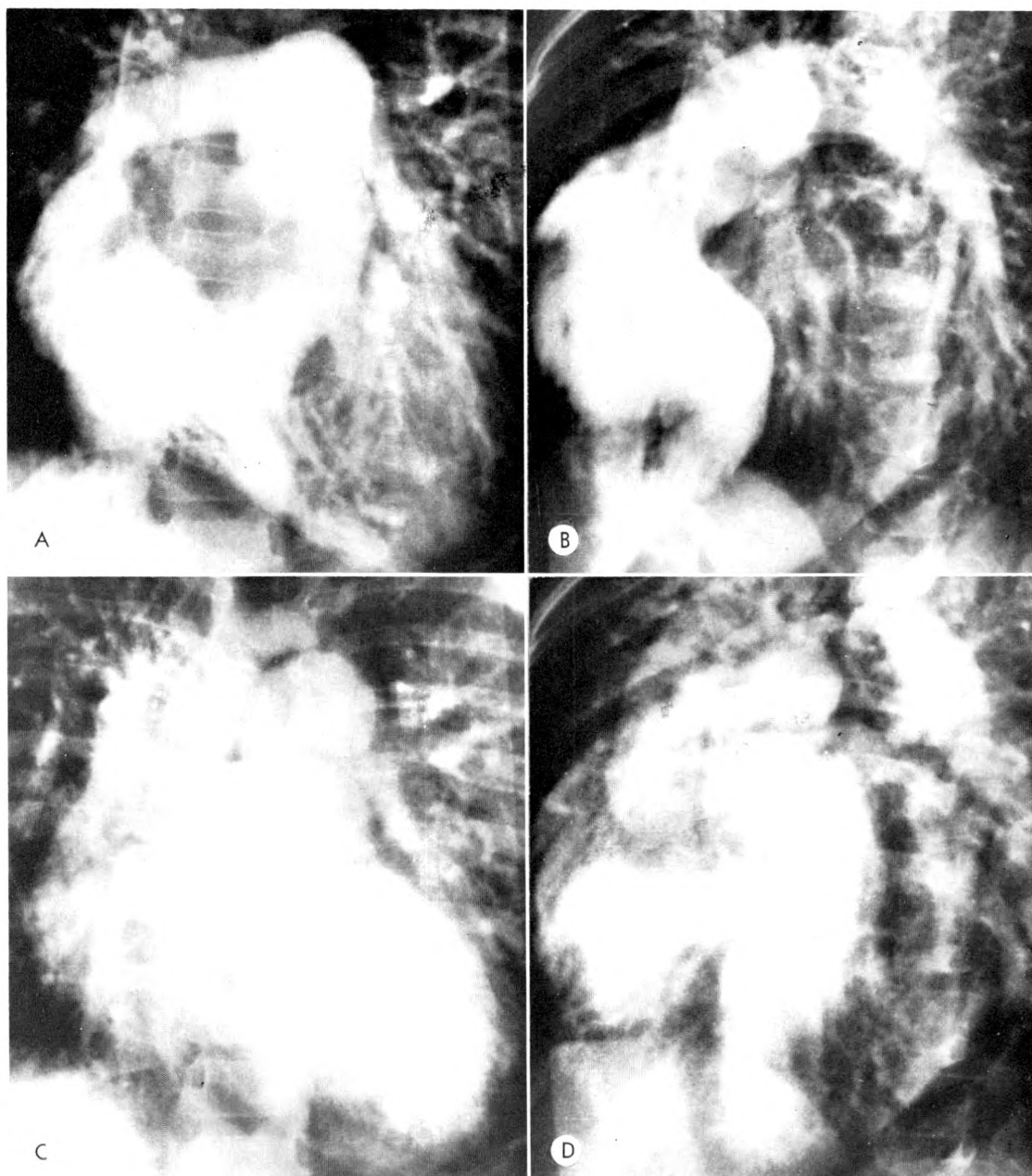


FIG. 7. Case 9. (A) Frontal intravenous angiogram showing enlargement of the right atrium, ventricle, pulmonary artery, and pulmonary vasculature. (B) Left anterior oblique angiogram also showing right ventricular and pulmonary arterial enlargement. (C) Frontal angiogram showing left atrial and ventricular enlargement with reopacification of the right ventricle and pulmonary arterial system. The aortic root is obscured by the opacified left atrium. The aortic arch is in the normal position. (D) The left anterior oblique angiogram, however, reveals the aortic root originating from the inflow tract of the right atrium. The base of the pulmonary artery and of the aorta is superimposed in this view.

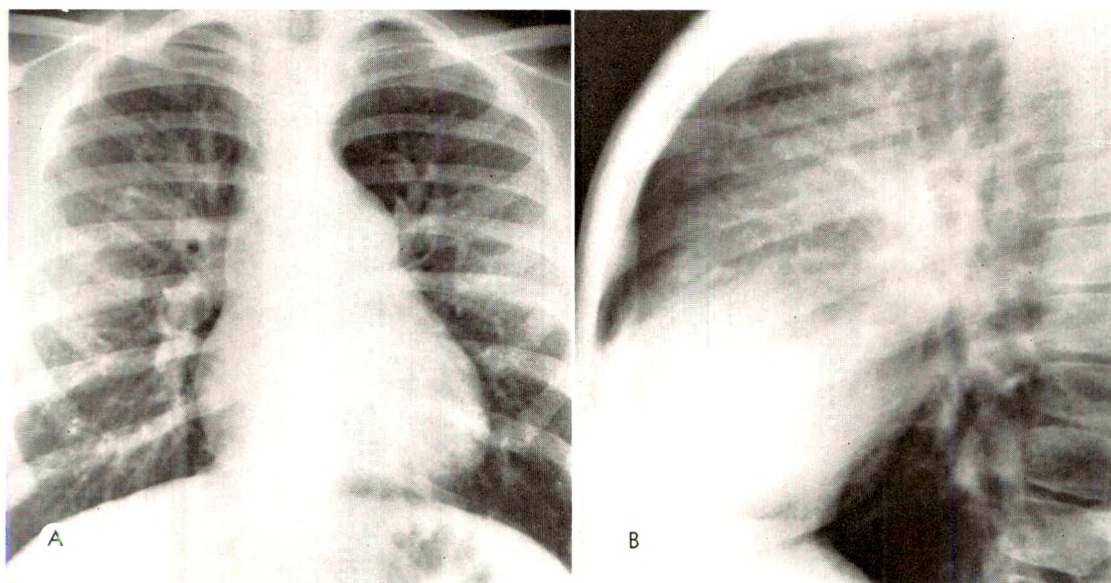


Fig. 8. Case 12. (A) Frontal teleroentgenogram of the chest showing enlargement of the heart and pulmonary artery. Although the central branches of the pulmonary artery are large, the peripheral branches are decreased. (B) Lateral roentgenogram of the chest showing anterior bowing of the sternum and enlargement of the right ventricle.

spective study. In a third case, the diagnosis of both great vessels arising from the right ventricle and pulmonic valvular stenosis was established by intravenous angiocardiology and verified at operation.

SUMMARY AND CONCLUSIONS

With the use of angiocardiology, the diagnosis of origin of both great vessels from the ventricle in association with a ventricular septal defect was established in 12 of 13 acyanotic patients. In the left anterior oblique view, and sometimes in the lateral view, an opacified stream of contrast material traversing the ventricular septal defect to the aortic root situated in the right ventricle is striking. This tunnel-like structure is best visualized during selective left ventriculography, although it has been observed following selective right ventriculography, and, rarely, following intravenous angiocardiology.

Patients with origin of both great vessels from the right ventricle cannot be differentiated from those with simple ventricular defects by clinical and conventional roent-

genographic findings. The correct diagnosis is important since a surgeon, not forewarned, may not see the aortic origin from the right ventricle and repair only the ventricular septal defect and cause a fatality.

Angiocardiology can also aid in differentiating other types of transposition of the great vessels from that of origin of both great vessels from the right ventricle. The Taussig-Bing anomaly can be recognized by the dense opacification of the right ventricular aorta and the faint opacification of the normally positioned or overriding pulmonary artery. Complete transposition of the great vessels can be demonstrated by showing that only the aorta arises from the right ventricle; origin of the pulmonary artery from the left ventricle may then be surmised or demonstrated if shunts permit. Corrected transposition can also be diagnosed by revealing that the aorta is the anterolateral vessel and the pulmonary artery lies posteromedially. Sometimes the appearance of the ventricles identifies them as inverted. Also, in physiologically corrected transposition, the medially placed pulmonary artery arises

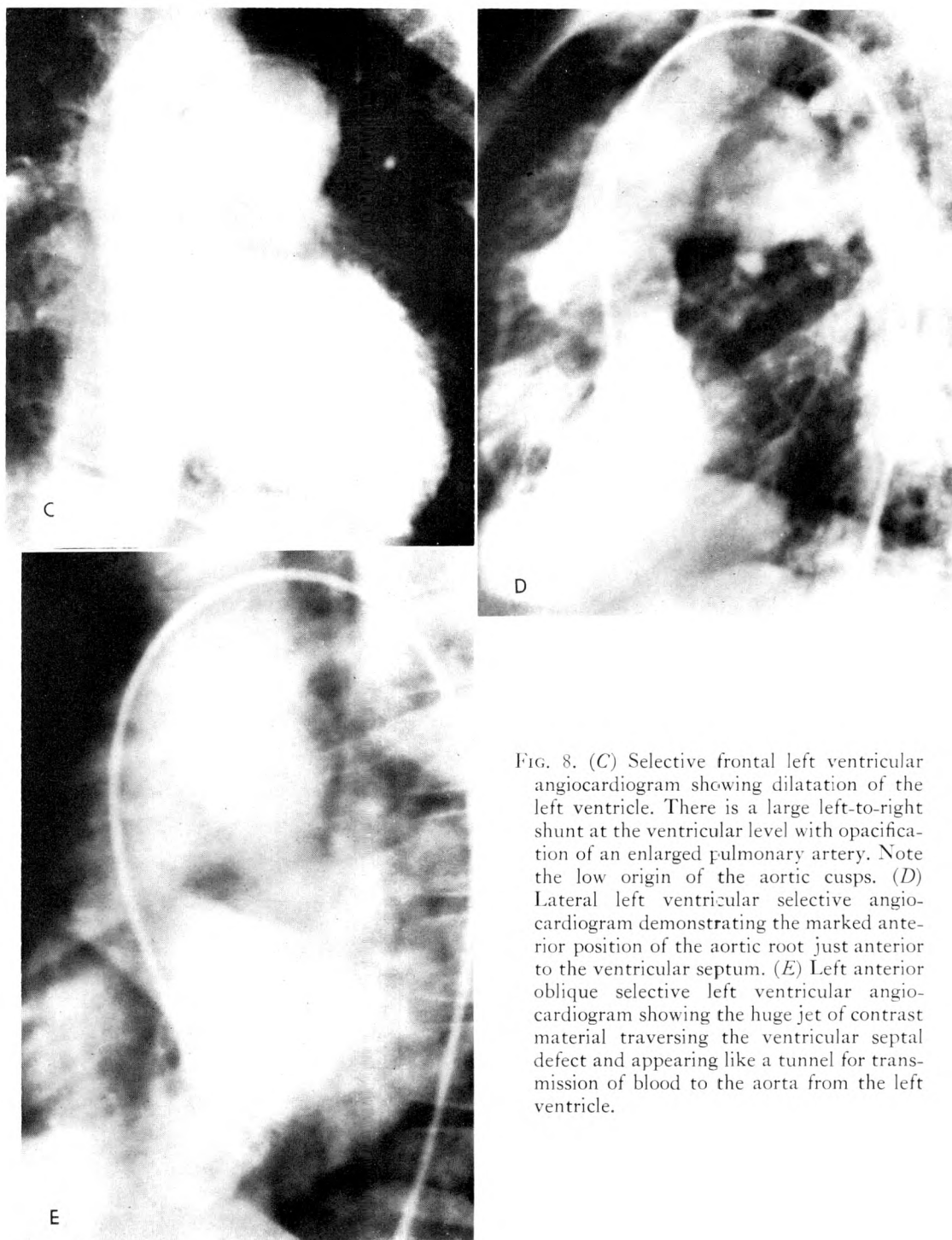


FIG. 8. (C) Selective frontal left ventricular angiogram showing dilatation of the left ventricle. There is a large left-to-right shunt at the ventricular level with opacification of an enlarged pulmonary artery. Note the low origin of the aortic cusps. (D) Lateral left ventricular selective angiogram demonstrating the marked anterior position of the aortic root just anterior to the ventricular septum. (E) Left anterior oblique selective left ventricular angiogram showing the huge jet of contrast material traversing the ventricular septal defect and appearing like a tunnel for transmission of blood to the aorta from the left ventricle.

from the venous ventricle, while the transposed aorta arises from the systemic ventricle along the left hilar border of the cardiac silhouette.

Cyanotic patients with both great vessels arising from the right ventricle and with pulmonic stenosis can also be differentiated from those with the tetralogy of Fallot by showing that the origins of both of the great vessels arise from the right ventricle.

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ADDENDUM

Since this paper was submitted for publication, an article on this subject was published in this JOURNAL by Cary, L. S., and Edwards, J. E. Roentgenographic features in cases with origin of both vessels from the right ventricle without pulmonary stenosis, 1964, 93, 269-297.

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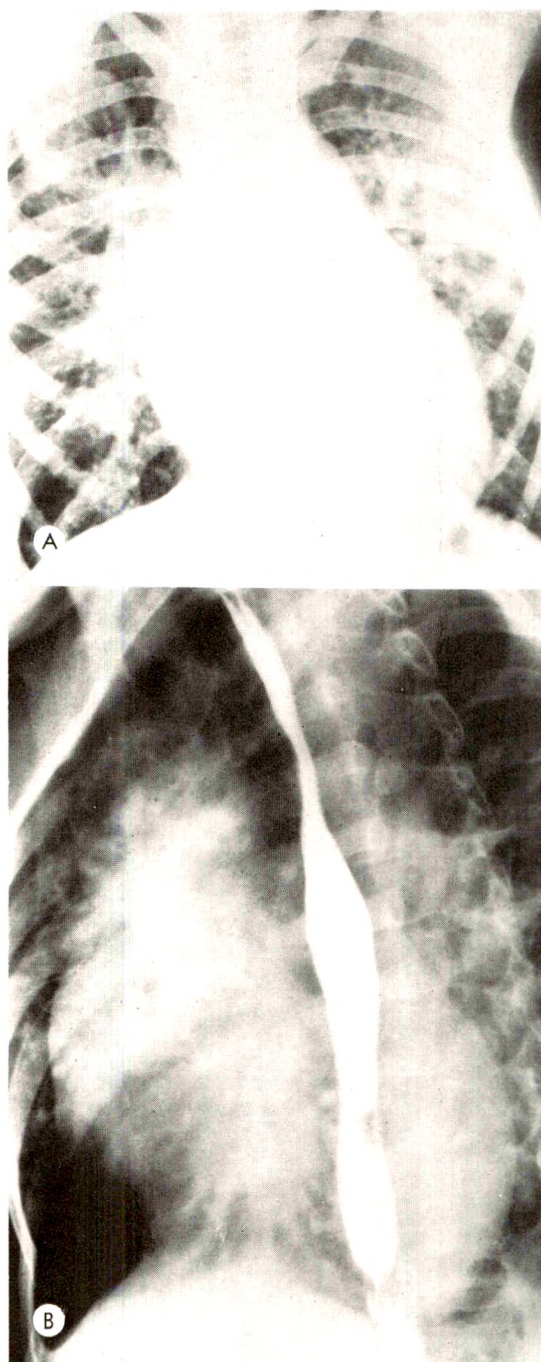


FIG. 9. Case 13. (A) Frontal teleroentgenogram of the chest showing marked enlargement of the heart and plethora of the lungs. (B) Left anterior oblique roentgenogram with esophagram demonstrating that both ventricles are enlarged.

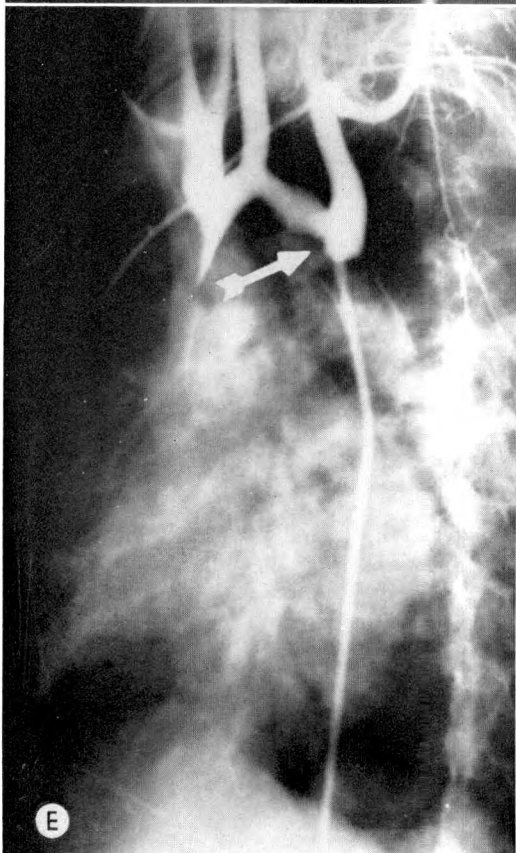
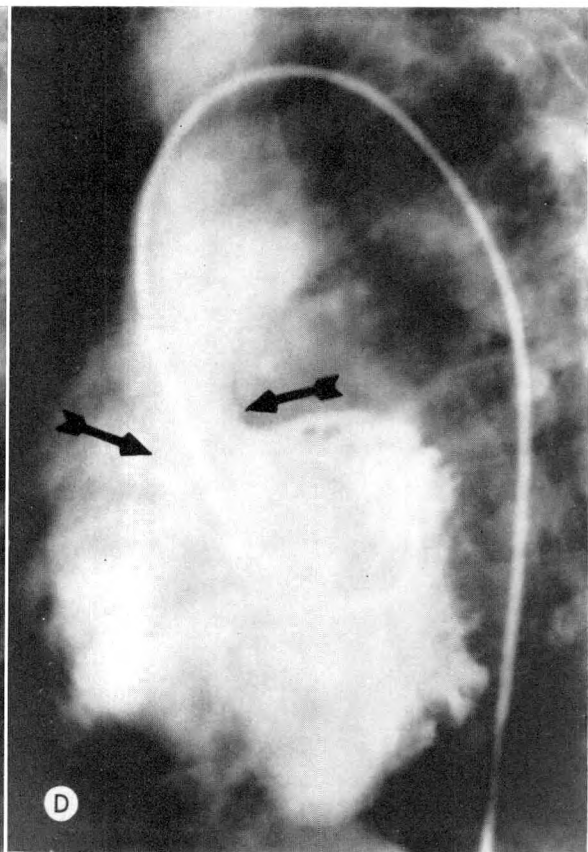
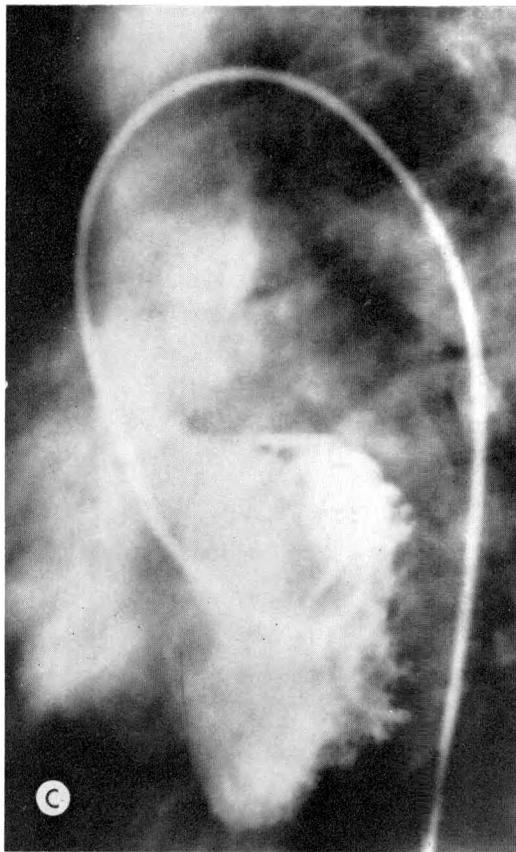


FIG. 9. (C) Left anterior oblique selective left ventricular angiogram showing the left-to-right shunt through the ventricular septal defect like a tunnel with the forward position of the aortic root arising from the right ventricle. (D) Later in the series, the tunnel-like shunt from the left ventricle to the right ventricle is again visualized (arrows). The posterior cusp of the aortic valve is anterior to the plane of the ventricular septum. The root of the aorta is above the opacified right ventricle. (E) Retrograde aortogram with catheter passed through the coarctated segment of the aorta demonstrating narrowing of the transverse aortic arch and a patent ductus arteriosus (arrow).

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ANOMALOUS SYSTEMIC VENOUS CONNECTION TO THE LEFT ATRIUM OR TO A PULMONARY VEIN*

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THE anomalously connected* systemic veins to the left atrium or a pulmonary vein include one or more of the following vessels: (1) left superior vena cava, (2) levo-atrio-cardinal vein including coronary sinus connecting to the left atrium, (3) right superior vena cava, (4) inferior vena cava, (5) azygos vein, and (6) hepatic vein. Our experience with 11 cases of this group of anomalies is presented. The practical significance of the preoperative diagnosis of these anomalies is emphasized.

EMBRYOLOGY

The paired precardinal veins are the drainage vessels for the primitive segments destined to become the head, neck and upper extremities. Paired postcardinal veins drain blood from the primitive caudal segments. These precardinal and postcardinal veins join to form two common cardinal veins (ducts of Cuvier) which in turn enter the common atrial chamber via an ante-chamber, the sinus venosus (Fig. 1A). This ante-chamber is a bicornuate structure into which drain the common hepatic and cardinal veins. As the right common cardinal and vitelline-umbilical veins assume greater functional significance, the right horn of the sinus venosus enlarges (Fig. 1B). The heart as a whole moves caudally into the thorax so that the common cardinal veins come to lie in the long axis of the embryo. With progressive

development, the sino-atrial region turns so that the left side comes to lie inferior to the right² (Fig. 1C). As this turning occurs, the right horn of the sinus venosus, as well as a portion of the transverse segment, becomes incorporated into the wall of the right atrium. Thus, the openings of the future superior vena cava and inferior vena cava are directly into the right atrium.

During the eighth fetal week, the right and left precardinal veins become connected by an oblique venous channel. This vein, the left innominate, under normal circumstances of development shunts blood from the left precardinal vein to the right precardinal vein (Fig. 1B). A portion of the blood from the left superior part of the body is therefore shunted to the right precardinal vein. The left precardinal vein between the left innominate and the left common cardinal vein becomes obliterated (Fig. 1C). The left horn of the sinus venosus and the partially obliterated left common cardinal vein, respectively, become the coronary sinus and the oblique vein of Marshall. The right precardinal and the right common cardinal veins become the superior vena cava.

The manner of development of the anastomotic vessel, the left innominate vein, between the precardinal veins in some cases seems to be significant in determining persistence of a left superior vena cava.³³ When this anastomotic vessel is small or nonexistent, the left superior vena cava persists. If the anastomotic vessel develops in its normal caliber but runs obliquely caudally from right to left, the right

* Connection does not necessarily indicate drainage. When the direction of blood flow is uncertain or other than normally anticipated, the term "connection" is used.

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superior vena cava may involute, leaving a left superior vena cava to drain the blood of the entire upper body. When the left superior vena cava persists, it usually drains into the right atrium via the coronary sinus.

Drainage of a persistent left superior vena cava into the left atrium is rare. When this occurs, there has been failure of turning of the sino-atrial region (Fig. 1, B and C) so that the left side lies at the same level as the right rather than in its normal inferior position.⁸ Thus, it can be seen that a persistent left superior vena cava draining into the left atrium is associated with absence of the coronary sinus. In addition, when bilateral superior venae cavae are present, an azygos vein joins each of them.⁸

Earlier case reports of persistent left superior vena cava connecting with the left atrium have not emphasized the presence or absence of a coronary sinus. Therefore, the true incidence of this type of abnormal connection cannot be evaluated.

Edwards and DuShane⁷ used the term levo-atrio-cardinal vein to indicate a collateral channel between the left atrium or pulmonary venous system and the cardinal venous system. This vein is embryologically different from the persistent left superior vena cava draining into the left atrium. In the early stages of development, the primordia of the respiratory system are seen to originate as a division of the foregut. At this stage the developing lungs share their blood supply with the foregut (Fig. 2) which is enmeshed by a capillary plexus, the splanchnic plexus.⁹ This plexus thus establishes direct communication between the pulmonary veins and the cardinal veins. As the lungs acquire a route of drainage into the left atrium, these communications are largely lost. If, however, at the time of partial incorporation of the pulmonary veins into the left atrium there were persistence of some of these early anastomoses, one might anticipate communication between a pulmonary vein or the left atrium and a derivative of the cardinal venous system.

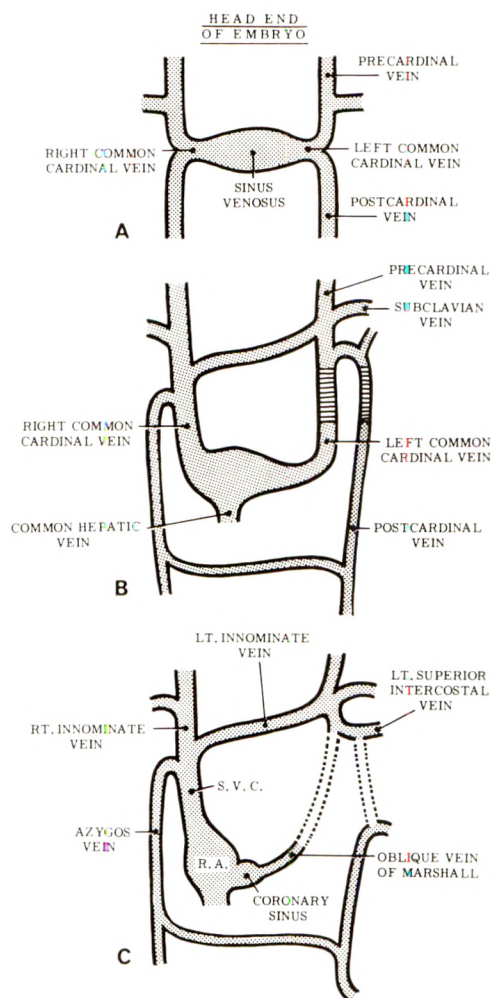


FIG. 1. (A, B and C) Normal transformation of the cardinal veins of the human embryo. (Redrawn from Campbell and Deuchar.²)

Persistence of a collateral channel has been described in cases of mitral atresia and narrow or closed foramen ovale where there is no normal outflow pathway from the left atrium.^{7,10,19} The levo-atrio-cardinal vein may then act as an escape route for oxygenated blood into a cardinal venous derivative, usually the left innominate vein.⁷ This situation is functionally a left-to-right shunt as opposed to the direct connection between a persistent left superior vena cava and the left atrium which yields a right-to-left shunt.

A levo-atrio-cardinal vein does not necessarily have a direct communication with

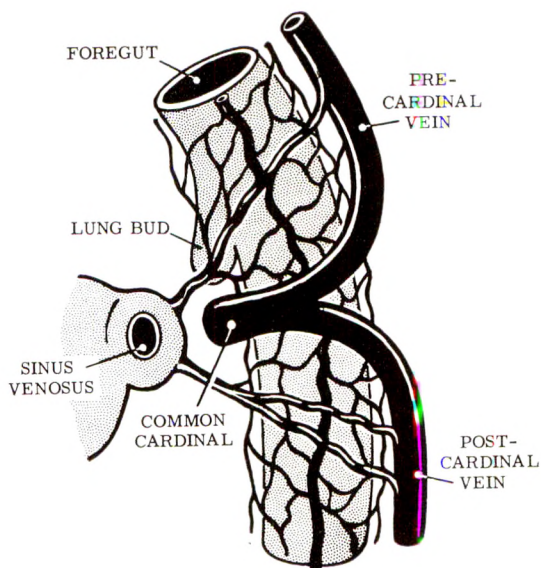


FIG. 2. The splanchnic plexus serves as an early communication between the future pulmonary veins and the future venae cavae. (Redrawn from Harris *et al.*¹⁵)

the left atrial chamber but may be present in form of a connection between a pulmonary vein and the left innominate vein.¹⁹ Edwards⁹ has also pointed out that a coronary sinus with a narrow termination in the right atrium may have a connection with the left atrium. In this instance, a primitive connection may have served as a collateral channel for coronary venous drainage into a left pulmonary vein early in development. When the pulmonary vein was partially incorporated into the left atrium, this channel may also have been effaced, permitting direct connection between the coronary sinus and the left atrium.⁹

The inferior vena cava has a complex embryologic history.²⁵ The only common anomaly of the suprarenal segment is that of azygos continuation. This has been described in the literature as "absence of the inferior vena cava" and "persistence of the supracardinal system." When the suprarenal segment of the inferior vena cava does not develop, the more caudal portion loses connection with the inferior aspect of the heart. The course for blood from the

infrarenal segment of the inferior vena cava is through the azygos vein to the right superior vena cava.¹⁷ If there is a left inferior vena cava present and the suprarenal segment fails to form, blood from the left inferior vena cava courses into the thorax via the left azygos vein. This latter vein may enter a persistent left superior vena cava after coursing over the left hilus. If the left superior vena cava enters the left atrium, blood from the caudal part of the body would be shunted to the systemic circulation.

CASE SUMMARIES

CASE 1. D.J., Hosp. No. 33-09-27. Two and one-half year old mildly cyanotic female who was always small for her age. A Grade 2/6 systolic ejection murmur was heard at the cardiac base. The preoperative diagnosis after cardiac catheterization and cinecardioangiography was ostium primum type atrial septal defect with mitral insufficiency. At the time of surgery, a large atrial septal defect was found and there was also a short cleft in the mitral valve leaflet. A persistent left superior vena cava, about the size of the right superior vena cava, entered the left atrium as did the pulmonary veins. An innominate vein connected the left and right superior venae cavae. The coronary sinus was absent. A teflon patch was used to separate the atria. The persistent left superior vena cava was ligated and the cleft mitral valve leaflet repaired. Two days after surgery, the patient died with acute pulmonary edema. Postmortem examination revealed that the hepatic vein entered separately, medial to the inferior vena cava into the right atrium.

CASE 2. C.F., Hosp. No. 33-26-66. Thirteen year old female who was asymptomatic. She was referred by the school physician because of an apical systolic heart murmur and thrill. No cyanosis was noted. The preoperative diagnosis after cardiac catheterization and cinecardioangiography was ostium primum type atrial septal defect with mitral insufficiency. A persistent left superior vena cava was seen draining into the left atrium (Fig. 4). At the time of surgery, the right superior vena cava was found to enter the right atrium. An innominate vein connected the superior venae cavae. Also a small ostium secundum type

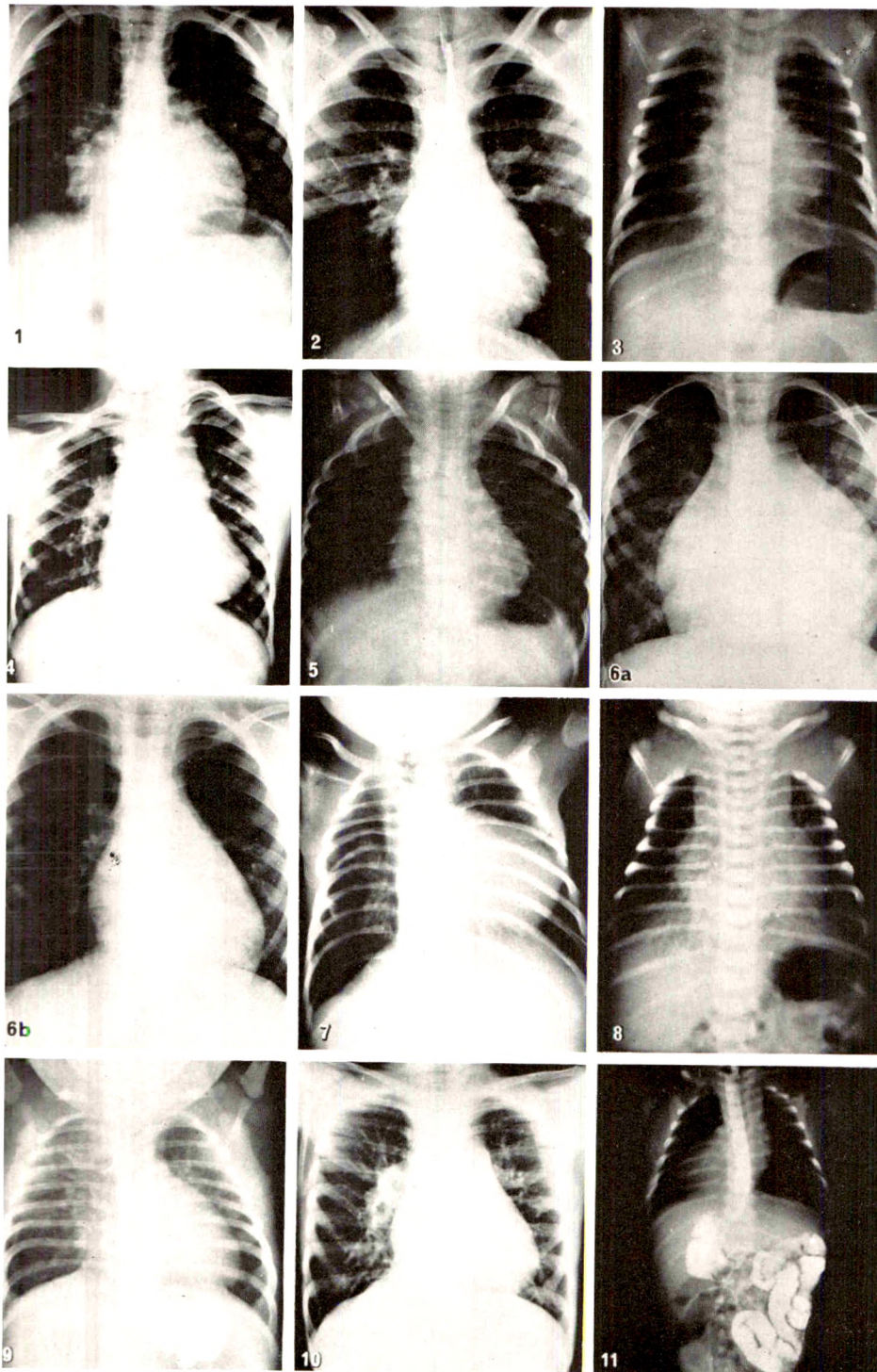


FIG. 3. A soft tissue density can be seen in the left upper mediastinum suggesting a persistent left superior vena cava in Cases 2, 4, 6 and 10. The marked decrease in cardiac size in Case 6 after reparative surgery is seen in 6a and 6b. The mild convexity noted along the left heart border in Case 10 slightly above the level of the left pulmonary artery corresponds to the left azygos vein as it courses over the hilus. Case 11 represents dextrocardia with the stomach and liver on the right. The proximal small intestine is to the left of the midline.

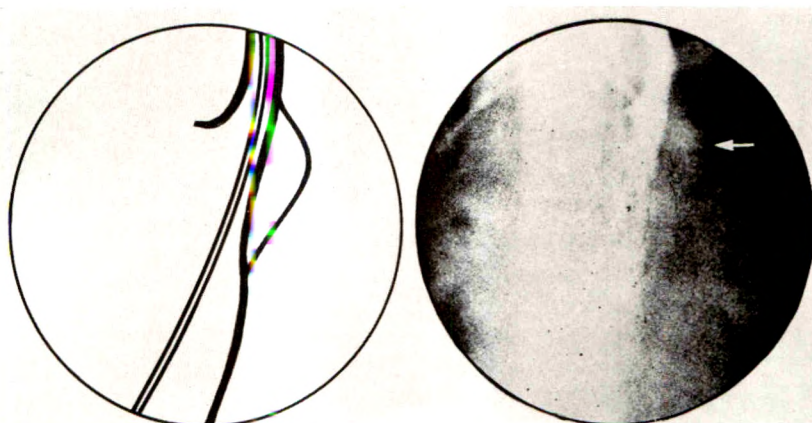


FIG. 4. Case 2. A cinerentgenographic frame with three catheters in place, one in the right pulmonary artery, one in the aorta and one in a persistent left superior vena cava having passed from the inferior vena cava through the right atrium, atrial septal defect, and left atrium. Injection into the left superior vena cava opacifies the left atrium. There is early opacification of the left atrial appendage (arrow).

atrial septal defect, as well as the primum defect, was present. The coronary sinus was absent. The atrial septal defects and cleft mitral valve leaflet were repaired. The left superior vena cava was ligated. The patient has done well postoperatively.

CASE 3. S.W., Hosp. No. 33-27-44. Six month old female who had frequent episodes of otitis media. A Grade 3/6 holosystolic murmur was heard along the left sternal border maximal at the third intercostal space. At the time of cardiac catheterization, the catheter passed into a left superior vena cava through what was thought to be the coronary sinus (Fig. 5). The postmortem examination showed tetralogy of Fallot with marked infundibular stenosis, a normal appearing pulmonary valve, and a patent foramen ovale. A persistent left superior vena cava was present between the left atrium and the left innominate vein. The right atrioventricular valve was bicuspid. The coronary sinus was absent. Additional anomalies in this patient included incomplete rotation of the colon, polycystic disease of the kidneys, and duplication of the cervix and vagina with a bicornuate uterus.

CASE 4. B.E., Hosp. No. 14-09-25. Nine year old female with mild cyanosis and shortness of breath on exertion. A loud systolic murmur was heard at the left third intercostal space. On September 29, 1950 cardiac catheterization was performed through the left antecubital vein. The catheter passed through a persistent left superior vena cava into what was thought to

be the coronary sinus and then into the right atrium (Fig. 6, A and B). Oxygen saturation data suggested the presence of an interatrial septal defect. In 1954 at the time of surgery, a persistent left superior vena cava was found entering the left atrium. The diagnosis of atrioventricularis communis was established at this time. The inferior vena cava and right superior vena cava entered the right atrium. Additional anomalies in this patient were mid-thoracic dextroscoliosis and hemivertebrae formation involving the second and third thoracic vertebrae.

CASE 5. J.D.C., Hosp. No. 26-61-48. Five and one-half year old male who was operated upon first at 4 days of age for an intestinal obstruction. Cyanosis was first noted at 4 months of age. Cardiac catheterization and cinecardioangiography indicated the diagnosis of tetralogy of Fallot. An end-to-side left subclavian to pulmonary artery anastomosis was performed when the patient was 14 months of age. Recently, the patient had reparative surgery. The shunt established earlier in life was surgically closed. A left superior vena cava entered the superior portion of the left atrium. The atrial septum was thought to be intact. A right ventricle infundibular obstruction was relieved and a defect in the ventricular septum was repaired. The left superior vena cava was transferred to the right atrium, medial to the entry of the right superior vena cava. The status of the coronary sinus is unknown.

Comment. Cases 1 through 5 represent

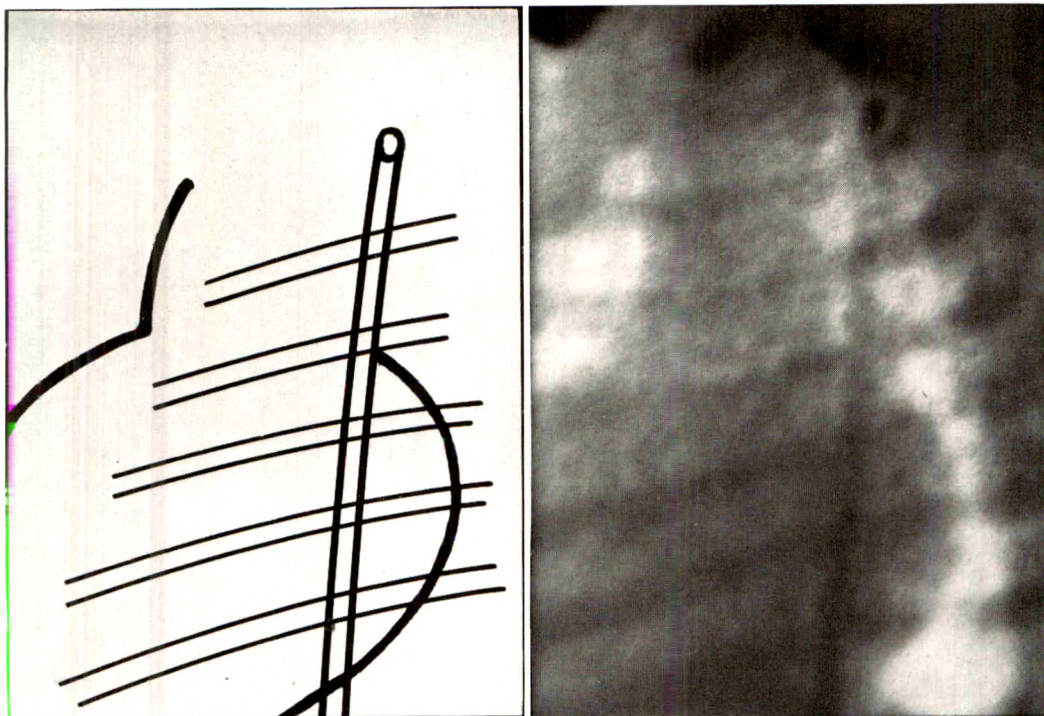


FIG. 5. Case 3. A cinerentgenographic frame which was thought to represent the catheter passing from the right atrium into a persistent left superior vena cava via the coronary sinus. This actually represents the catheter traversing the atrial septal defect and entering the persistent left superior vena cava via the left atrium. Film exposed in the left anterior oblique projection.

examples of persistent left superior vena cava draining into the left atrium. The right superior vena cava was present in all cases and drained into the right atrium. The coronary sinus was absent in 3 cases. Information regarding its presence or absence is not available in Cases 4 and 5.

CASE 6. K.P., Hosp. No. 33-12-84. Four year old female in "good health" until 3 weeks prior to admission when she developed mumps. At that time episodes of dyspnea and orthopnea were noted. No cyanosis or clubbing was present. A Grade 3/6 apical holosystolic murmur and a grade 3/6 mid diastolic murmur were heard over the cardiac apex. Cardiac catheterization and cinecardioangiography indicated absence of the right superior vena cava and drainage of a persistent left superior vena cava into the left atrium (Fig. 7 and 8). In addition there was evidence of an ostium primum type atrial septal defect and mitral insufficiency. At the time of surgery, a common atrium or a large atrial septal defect was found. The right superior vena cava was absent. The inferior

vena cava was in normal position. The right innominate vein joined the left innominate vein and descended as a left superior vena cava which entered the left atrium. No coronary sinus was present. The atria were partitioned with a teflon patch and a cleft in the mitral valve leaflet was repaired. The left superior vena cava was transferred to the right atrium. Postoperatively the patient did well with roentgenographic evidence of marked reduction in heart size (Fig. 3, 6a and 6b).

Comment. In this case the persistent left superior vena cava served as the only final pathway for venous drainage for the head and upper body, the right superior vena cava being absent.

CASE 7. J.M., Hosp. No. 26-38-97. Male seen at 2 days of age with esophageal atresia, tracheo-esophageal fistula and duodenal atresia. A Grade 3/6 systolic murmur was heard over the left third intercostal space. Postmortem examination 5 weeks after birth revealed tricuspid valve atresia, ostium secundum type atrial

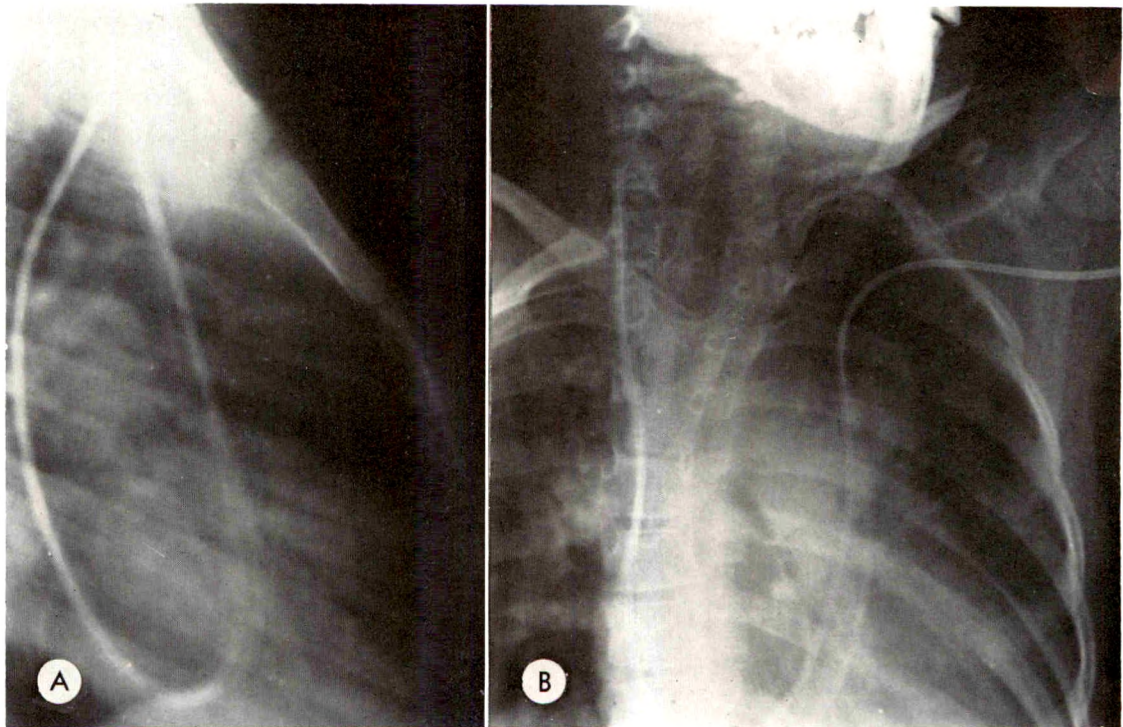


FIG. 6. Case 4. (A and B) Spot roentgenograms of the catheter passing from the left antecubital vein into a persistent left superior vena cava. The course was thought to be through the coronary sinus and the right atrium into the right superior vena cava. At the time of surgery the persistent left superior vena cava was found to enter the left atrium.

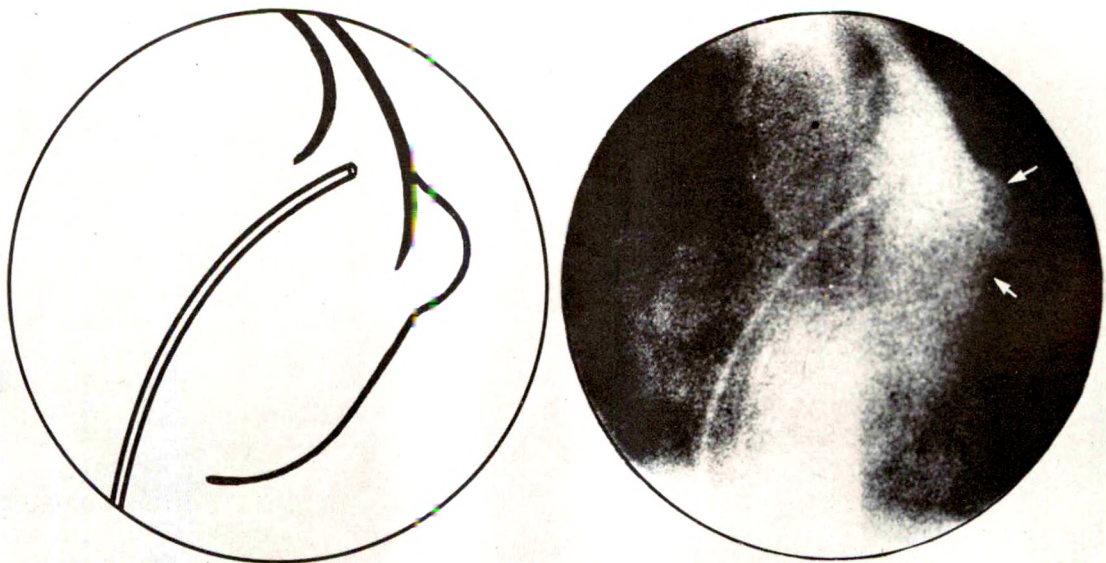


FIG. 7. Case 6. A cinerentgenographic frame with the catheter in the superior portion of the left atrium at the



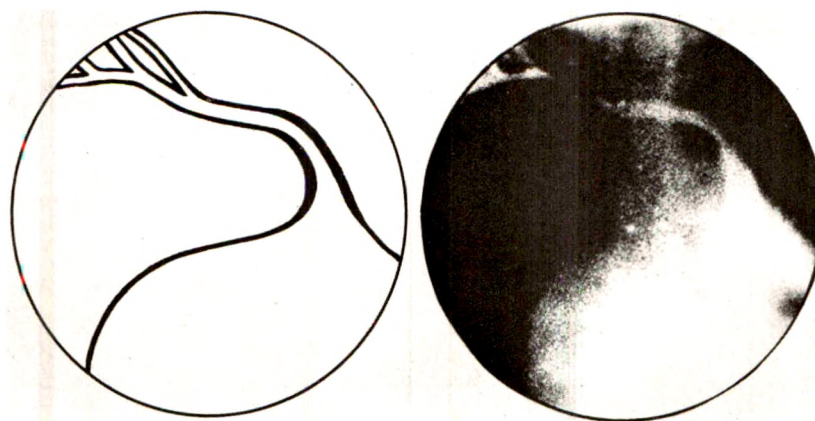


FIG. 8. Case 6. A cinerentgenographic frame with an injection in the right brachial vein. There is opacification of the innominate vein and the persistent left superior vena cava which in turn enters the left atrium. The left atrium and atrial appendage were opacified before the right atrium, excluding drainage of the persistent left superior vena cava into the coronary sinus. The right superior vena cava is absent.

septal defect, hypoplastic right ventricle and pulmonary artery, and a right aortic arch. An anomalous vessel connecting a cervical vein to the left atrium was found. The pulmonary veins entered the left atrium in a normal fashion. The coronary sinus was present.

CASE 8. B.S., Hosp. No. 24-02-92. One week old cyanotic male with a Grade 2/6 systolic murmur heard along the left sternal border. The patient died 2 weeks after birth in severe congestive heart failure. Postmortem examination revealed coarctation of the aorta between the left common carotid and the left subclavian artery, bicuspid aortic valve, atresia of the mitral valve, hypoplasia of the left ventricle, ventricular septal defect, patent ductus arteriosus, and an unusual venous anomaly. There was a vein connecting the innominate vein and a left pulmonary vein. An additional connection between the anomalous vein and a smaller vein which coursed along the left side of the vertebral column was noted (Fig. 9).

Comment. Cases 7 and 8 represent examples of a levo-atrio-cardinal vein associated with other complex cardiovascular

anomalies. In Case 7 without mitral atresia, this vein served as a direct connection between the left atrium and a derivative of the cardinal system. In Case 8 with mitral valve atresia and absence of a defect in the atrial septum, the levo-atrio-cardinal vein served as the only drainage pathway for pulmonary venous blood from the left atrium. Even though this persistent collateral channel did not connect directly to the left atrium, its developmental and functional roles justify usage of the term levo-atrio-cardinal vein.

CASE 9. R.C., Hosp. No. 21-79-90. Three month old male who was first seen by his physician because of a growth on the tongue. At the time of this examination, a loud systolic murmur was heard along the left sternal border at the third and fourth intercostal spaces. No cyanosis or clubbing was present. At the time of cardiac catheterization, the catheter was introduced into the right saphenous vein and went to the left of the midline entering the heart at the level of the left atrium. Angiocardiography was performed via the left femoral

site of entry of the persistent left superior vena cava. The catheter passed from the inferior vena cava into the right atrium and through the atrial septal defect into the left atrium. An injection of contrast medium at this site outlined the funnel configuration representing opacification of the superior portion of the left atrium and the entrance of the persistent left superior vena cava. The left atrial appendage (arrows), which is anterior, was better opacified on subsequent frames when the opaque medium filled the entire atrium. The two other catheters are in the right atrium and aorta.

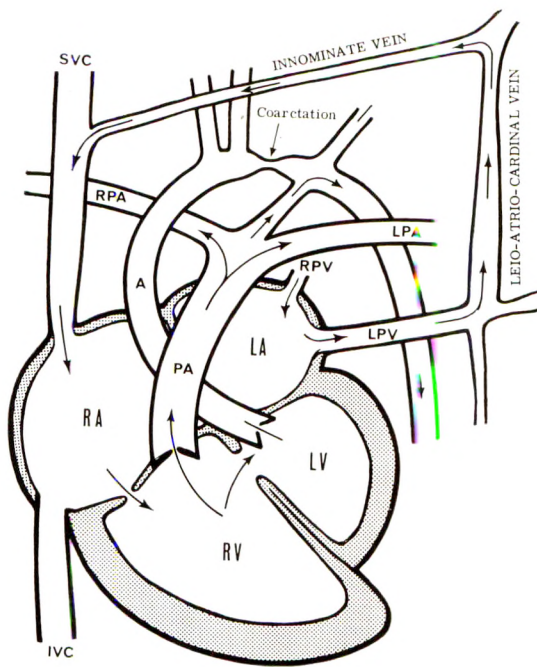


FIG. 9. Case 8. Diagram (not drawn to scale) of the heart and great vessels as studied at postmortem examination. Coarctation of the aorta, mitral valve atresia, hypoplasia of the left ventricle, ventricular septal defect and the levo-atrio-cardinal vein were found. SVC—superior vena cava; IVC—inferior vena cava; RPA—right pulmonary artery; LPA—left pulmonary artery; RPV—right pulmonary vein; LPV—left pulmonary vein; RA—right auricle; LA—left auricle; RV—right ventricle; LV—left ventricle; PA—pulmonary artery; A—aorta.

vein (Fig. 10, *A* and *B*). Later during the hospital course, the patient developed severe diarrhea and rectal bleeding. This did not respond to therapy and the patient died after 4 months of life. Postmortem examination indicated the presence of an anomalous left inferior vena cava with continuation into the thorax as the left azygos vein. The latter vein arched anteriorly over the hilus and joined the left superior vena which, in turn, drained into the left atrium. A right superior vena cava drained into the right side of the common atrium. There was absence of the interatrial septum, a small interventricular septal defect, a common atrio-ventricular valve, and a bicuspid stenotic pulmonary valve. Other significant findings were ileitis and ulcerative ascending colitis, fibromata of the tongue, syndactylism and polydactylism. The hepatic veins joined to

form a single vessel which entered the right atrium. A single spleen weighing 15 gm. was present.

CASE 10. J.C., Hosp. No. 34-49-24. Eight year old male with moderate cyanosis since the age of 4. He had very limited exercise tolerance and poor growth. A Grade 3/6 prolonged systolic ejection murmur and a systolic ejection click were heard along the left sternal border. Cardiac catheterization and cineangiocardiology were performed. The venous catheter was introduced into the right femoral vein and coursed upward to the left of the midline slightly above the level of the left hilus. It then passed anteriorly, entering the left atrium and then to the right atrium and right superior vena cava (Fig. 11*A*). Injection of contrast medium into the left azygos vein opacified this vessel and outlined its course over the left hilus to join the persistent left superior vena cava (Fig. 12, *A* and *B*). On one occasion, the catheter entered the hepatic vein from the right atrium. Because the right ventricle could not be entered from the femoral vein catheterization, a second study was done 24 hours later. The right antecubital vein was catheterized and the catheter entered the right atrium and ventricle via the right superior vena cava. The catheter could also be passed from the right atrium to the left atrium into the persistent left superior vena cava (Fig. 11*B*). The diagnosis after these examinations was anomalous left inferior vena cava with continuation into the thorax as the left azygos vein. The latter vessel entered the left atrium after joining the persistent left superior vena cava. Postmortem examination after cardiac surgery revealed a common atrium with the right superior vena cava and hepatic veins draining into the right side of the atrium. The persistent left superior vena cava after being joined by the left azygos vein entered the left side of the atrium. There was absence of the coronary sinus and a cleft in the anterior mitral valve leaflet. Twelve small splenic masses were noted in the region usually occupied by the spleen. An accessory lobe of the liver was found.

Comment. Cases 9 and 10 are examples of anomalous left inferior vena cava with left azygos continuation into the thorax. This azygos vein drains into a persistent left superior vena cava which in turn enters the left atrium.

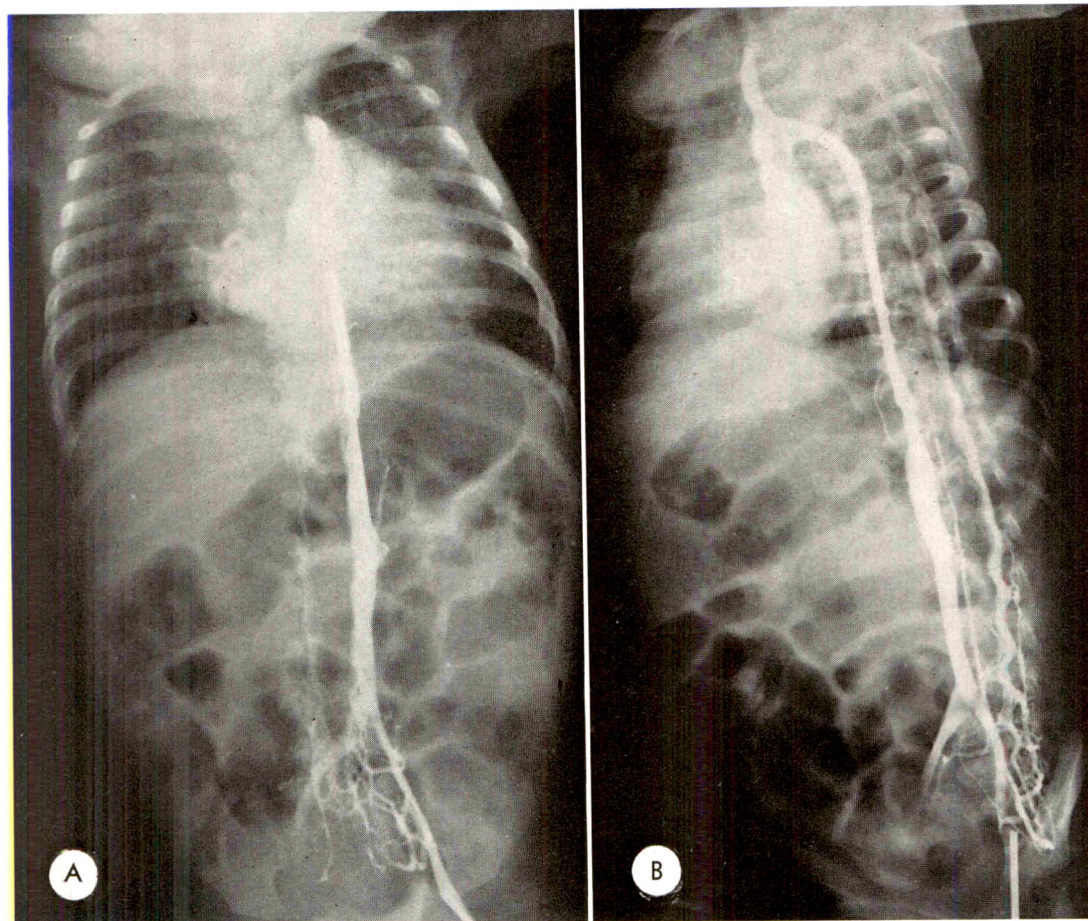


FIG. 10. Case 9. (A and B) Angiocardiograms with the injection of contrast medium into the left femoral vein. The anomalous left inferior vena cava with left azygos continuation into the thorax is opacified. The left azygos vein is seen coursing over the left hilus and joining the persistent left superior vena cava. The latter vessel is seen entering the left atrium.

CASE 11. M.A., Hosp. No. 35-10-63. Deeply cyanotic 6 week old male. A loud systolic heart murmur was heard over the right chest. Cardiac catheterization and cinecardioangiography were performed. The catheter was introduced into the right femoral vein and ascended in the abdomen to the left of the spine. Before entering a posterior atrium, it crossed to the right of the spine. Postmortem examination revealed absence of the spleen and partial transposition of the abdominal viscera. There were 3 lobes present in both lungs. The cardiac malformation consisted of large atrial and ventricular septal defects, mixed dextrocardia with mirror image ventricles and pivoted atria, atrioventricularis communis, transposition of the great vessels, pulmonary atresia, patent ductus arteriosus, total anomalous pulmonary venous

drainage to the left superior vena cava which entered the systemic (left) atrium, absence of the coronary sinus, and absence of the right superior vena cava (Fig. 13).

Comment. Case 11 represents an example of complex cyanotic congenital heart disease associated with asplenia and abdominal heterotaxia. The left superior vena cava drained anomalously into the left (systemic) atrium after receiving the pulmonary veins. The right superior vena cava was absent.

DISCUSSION

A. *Persistent Left Superior Vena Cava Draining into the Left Atrium Associated with Other Cardiac Anomalies.* The presence of a

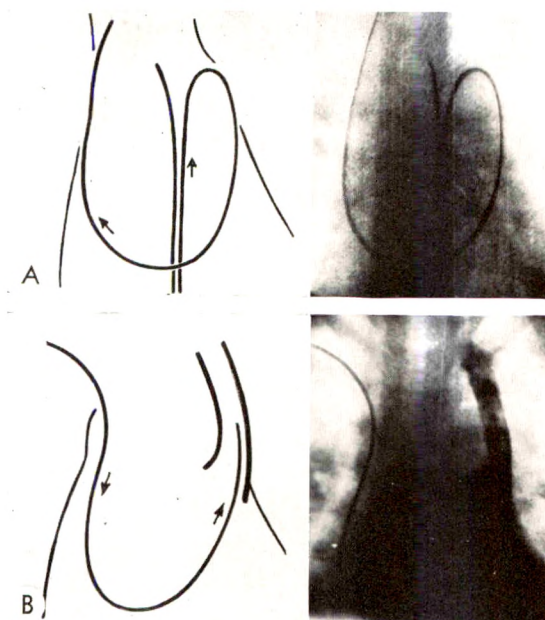


FIG. 11. Case 10. (A) A cinerentgenographic frame with the straight catheter in the aorta. The curved catheter was introduced through the right femoral vein and coursed to the left of the midline. It passed upwards into a persistent left superior vena cava via an anomalous left inferior vena cava with left azygos continuation. From the persistent left superior vena cava, it coursed through the left atrium, atrial septal defect and right atrium to the right superior vena cava. (B) The catheter was passed from the right antecubital vein into the right atrium, left atrium, and then into the persistent left superior vena cava. An injection into this vessel indicates drainage into the left atrium.

left superior vena cava is not usually appreciated during analysis on plain roentgenograms of the cardiovascular system. Frontal roentgenograms of the chest may indicate widening of the left upper mediastinum, and, occasionally, a separate shadow³³ may suggest the presence of this vessel. The drainage of a persistent left superior vena cava usually occurs via the coronary sinus into the right atrium. In 10 of 174 cases of persistent left superior vena cava collected by Winter,³³ drainage occurred into the left atrium and all of these patients had intracardiac septal defects. In another 11 of this group of 174 cases, the left superior vena cava connected with a common atrium or with a persistent sinus venosus.

In patients with partial transposition of the viscera, there is a significant incidence of persistent left superior vena cava draining into the left atrium.^{2,3,12,33} In 5 of 47 patients with persistent left superior vena cava reported by Campbell and Deuchar,² this vessel communicated with the left atrium. Three of these 5 cases had partial transposition of the viscera. In 15 of a group of 17 cases with a syndrome of congenital cardiac disease and asplenia reported by Ruttenberg *et al.*,²⁷ bilateral superior venae cavae with absent coronary sinus were present. In most of these patients, abdominal heterotaxia (*i.e.*, right sided stomach and a nearly symmetric liver) was present.

The demonstration of the site of communication of the persistent left superior vena cava does not necessarily indicate the direction of the blood flow. This is shown

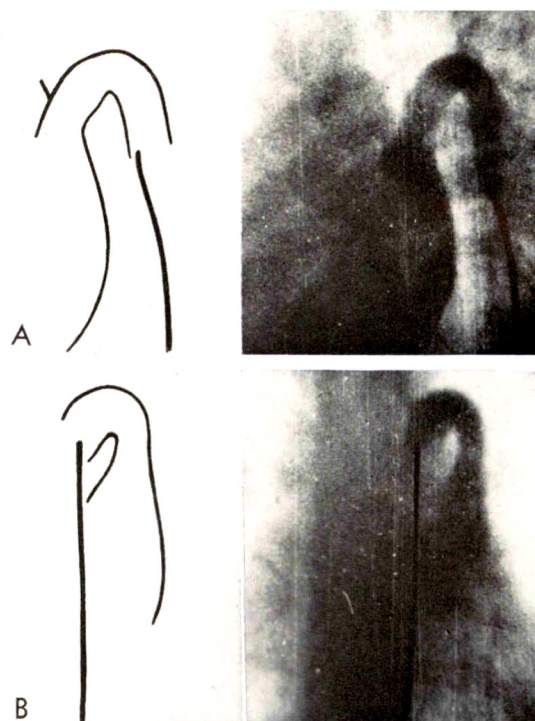


FIG. 12. Case 10. Cinerentgenographic frames with the catheter in the left azygos continuation of an anomalous left inferior vena cava. The contrast medium outlines azygos drainage into a persistent left superior vena cava which in turn enters the left atrium. (A) Left anterior oblique projection. (B) Posteroanterior projection.

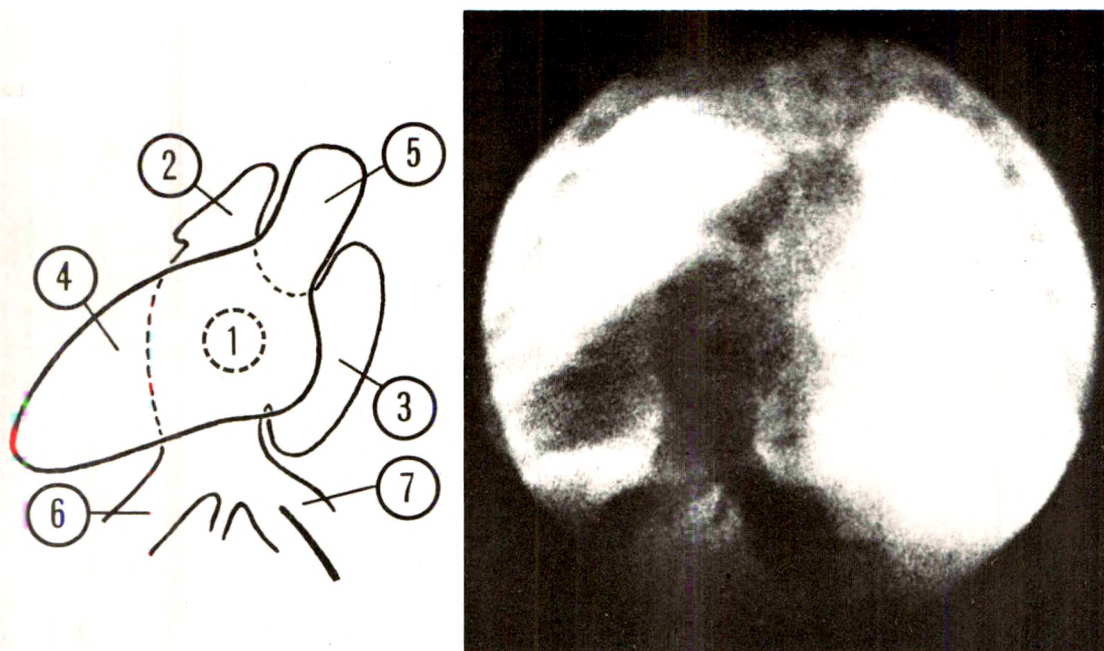


FIG. 13. Case 11. A cinerentgenographic frame with the catheter in the anomalous inferior vena cava. There is mixed dextrocardia present with mirror image ventricles and pivoted atria. The inferior vena cava (7) is opacified and drains into the pulmonary (right) atrium which is posterior (1). Some opaque medium has passed into the left ventricle (4) and the systemic (left) atrium (3). The appendage of the posterior (right) atrium is opacified (2). There is reflux opacification of the hepatic vein (6) and aortic opacification (5).

by the case reported by Ödman;²⁴ a persistent left superior vena cava communicated with a pulmonary vein and the left atrium. The contrast medium injected into the pulmonary artery, upon returning from the pulmonary circulation, opacified the left superior vena cava, the innominate vein and right superior vena cava.

In cases with two superior venae cavae, a communication between the right and left in the form of an innominate vein may or may not be present.^{26,33}

In patients with a persistent left superior vena cava draining into the left atrium, one or more of the pulmonary veins may drain into the left atrium, right atrium, right superior vena cava, left superior vena cava, innominate vein, sino-atrial chamber, or a common atrium.³³

Cardiac anomalies reported in patients with a persistent left superior vena cava communicating with left atrium are: atrial septal defect,^{12,22,33} single atrium^{2,11,33} ventricular septal defect,^{2,11,14,33} single ven-

tricle,^{2,33} tetralogy of Fallot,^{2,5,6,12,17,33,34} patent ductus arteriosus,^{14,26} pulmonary stenosis and atresia,^{12,14,16,31,34} truncus arteriosus,^{14,33} transposition of the great vessels,^{2,34} coarctation of the aorta,^{24,26} dextrocardia,^{2,3} and inferior vena caval anomalies.^{6,14,29,31,34}

B. Communication of Persistent Left Superior Vena Cava and Coronary Sinus with Both Left and Right Atria. The persistent left superior vena cava before reaching the right atrium courses onto the surface of the left atrium and has a connection with the left atrium.^{20,22,28} In a case reported by McCotter,²⁰ the left superior vena cava became dilated in its terminal portion and an oval foramen connected this vessel and the left atrium. Mankin and Burchell's²² case is of special interest. The right pulmonary vein drained into the left superior vena cava which in turn emptied into the coronary sinus. This was hemodynamically largely corrected by an anomalous opening between the coronary sinus and left atrium.

C. *Levo-Atrio-Cardinal Vein.* This group includes an anomalous connection between the left atrium or a pulmonary vein and any derivative of the cardinal venous system.⁷ Mitral atresia with a narrow or closed foramen ovale may be associated with the persistence of a collateral communication between the left atrium and the cardinal venous system.^{7,10,19,21} This persistent collateral vein serves as a drainage pathway for pulmonary blood in the absence of any other exit from the left atrium. However, in a case reported by Harris *et al.*¹⁵ under the name "jugulopulmonary vein," the persistent collateral vessel connected the left atrium to the right internal jugular vein in the presence of a patent mitral valve. The opening of the coronary sinus into the right atrium was atretic in this patient and it drained through a persistent left superior vena cava into the left innominate vein.

A levo-atrio-cardinal vein may connect a pulmonary vein and a systemic vein as seen in a patient reported by Lucas *et al.*¹⁹ The angiographic examination of this patient demonstrated the pulmonary venous drainage through the collateral vein to the innominate vein, right superior vena cava, and finally to the right atrium. This patient had an atretic mitral valve.

As indicated under embryology, communication of the coronary sinus and left atrium may be considered a form of levo-atrio-cardinal vein. In this situation the opening of the coronary sinus into the right atrium may be stenotic or atretic.¹⁰ Communication of the coronary sinus and left atrium as an isolated anomaly is of no clinical significance.²⁰ Associated anomalies may be an anomalous connection of the pulmonary vein to the left innominate vein¹⁰ or persistent left superior vena cava connected to the right pulmonary vein.³⁰

D. *Drainage of a Major Systemic Vein into the Left Atrium As an Isolated Anomaly.* Diagnosis of this type of anomaly^{4,13,18,23,32} is very important because of the excellent surgical results obtainable.¹⁸ Cyanosis out of proportion to the apparent physical and

cardiac status of the patient, left ventricular enlargement, and lack of roentgen findings compatible with other forms of cyanotic heart disease should make the correct diagnosis possible.^{4,18} This anomaly is compatible with full cardiac competence until middle age.²³ Davis *et al.*⁴ reported a case of a persistent left superior vena cava draining into the left atrium. Kirsch *et al.*¹⁸ described a right superior vena cava draining into the left atrium which was successfully corrected by surgical transposition of the anomalous vessel to the right atrium. The angiocardiographic examination of this patient with a left antecubital vein injection of contrast medium demonstrated anomalous drainage of the right superior vena cava into the left atrium. With a right ventricular injection, the upper pulmonary vein was seen to enter the left atrium in a more caudad position than normal and the contrast medium reaching the aorta was more diluted than usually anticipated. In the patient of Tuchman *et al.*,³² with anomalous drainage of a persistent left superior vena cava into the left atrium, a diagnosis of polycythemia rubra vera was entertained before cardiac catheterization. Gardner and Cole's¹³ patient with inferior vena caval drainage into the left atrium survived to adult life. Another case with a similar anomaly was described by Meadows *et al.*²³

E. *Drainage of Both the Superior Vena Cava and Inferior Vena Cava into the Left Atrium.* This is a rare anomaly which may be associated with small right ventricle, atresia of the pulmonary artery, and tricuspid atresia.³¹

F. *Azygos Vein and/or Hepatic Vein Draining into the Left Atrium.* In the absence of the suprarenal portion of the inferior vena cava, the azygos veins may become major sources of drainage of blood from the lower half of the body. In 2 of our patients (Cases 9 and 10) the left azygos after joining a persistent left superior vena cava drained into the left atrium. The hepatic vein may enter a common atrium close to the pulmonary veins.³¹

PRACTICAL SIGNIFICANCE OF THE
DIAGNOSIS OF THESE ANOMALIES

1. Drainage of a systemic vein into the left atrium as an isolated anomaly producing a right to left shunt is amenable to surgical correction.

2. Diagnosis and surgical management of anomalous systemic venous drainage as a complicating factor of other congenital cardiac lesions would improve the condition of the patient and tend to decrease cyanosis.^{5,11,16} In a patient reported by Diaz and Anido⁵ with tetralogy of Fallot and anomalous systemic venous drainage following closure of the left superior vena cava-left atrial connection, the hematocrit dropped from 91 per cent to 56 per cent. In Feindt and Hauch's¹¹ case with Eisenmenger's syndrome, the oxygen saturation increased from 70 per cent to 84 per cent following ligation of the connection between the left superior vena cava and the left atrium.

3. In a patient with either single atrium, large atrial septal defect particularly of the ostium primum type with mitral insufficiency, anomalous inferior vena cava, isolated levocardia with situs inversus abdominalis, or the syndrome of visceral symmetry with asplenia, a careful search for a persistent left superior vena cava-left atrial connection should be made. In our series of 9 cases of persistent left superior vena cava-left atrial communication, 7 had large atrial septal defects. In 4 cases mitral insufficiency was demonstrated by cine-cardioangiography. If during cardiac catheterization in a patient with atrial septal defect the catheter enters the left superior vena cava from what is considered to be the right atrium, angiocardiology in frontal and lateral projections is recommended to be certain that the vessel does not enter the left atrium.

4. Recognition of the anomalous systemic venous connection does not necessarily reveal the direction of blood flow. A pulmonary artery injection of contrast material may bring about opacification of the left atrium from the pulmonary veins.

The anomalous vessel may then become opacified, indicating flow away from the heart to the systemic circulation.

5. Knowledge of the presence of a persistent left superior vena cava and its drainage site is essential in planning cannulations for cardiopulmonary bypass, and in planning superior vena cava-pulmonary artery anastomotic procedures.

6. In patients with a primum type atrial septal defect and persistent left superior vena cava, the funnel shape entrance of this vessel may extend to the site of the defect in the septum. With an absence of the anterior wall of the funnel, the posterior aspect may form a part of the posterior wall of the left atrium. Recognition of this complicated type of atrial septal defect is of importance.

SUMMARY

Different types of anomalous systemic venous connection to left atrium or to a pulmonary vein are described and the developmental origin of some of these anomalies is discussed.

Drainage of a systemic vein into the left atrium as an isolated anomaly can be suspected clinically and proven by cardiac catheterization and angiocardiology. Such a lesion is amenable to complete surgical correction. The recognition of these anomalies in conjunction with other congenital heart lesions is of practical importance in the surgical management of these patients.

The association of persistent left superior vena cava-left atrial connection with an ostium primum type atrial septal defect and absence of the coronary sinus is emphasized.

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INFUNDIBULAR OBSTRUCTION SECONDARY TO PULMONARY VALVULAR STENOSIS*

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OBSTRUCTION of the right ventricular outflow tract is seen in tetralogy of Fallot and also in certain cases of pulmonary valvular stenosis with an intact ventricular septum. It is the purpose of the authors to show that these diseases differ significantly in the pathophysiology of the infundibular obstruction and that this knowledge may be useful in the surgical management of patients with infundibular obstruction secondary to pulmonary valvular stenosis.

Tetralogy of Fallot represents a primary form of outflow tract obstruction caused by developmental anomalies. In pulmonary valvular stenosis with an intact ventricular septum, on the other hand, contraction of hypertrophied muscle bundles may narrow the outflow tract, causing a secondary form of obstruction. There is evidence that in some but not all cases of secondary infundibular hypertrophy there is resolution of the infundibular narrowing following valvulotomy.

It is essential that those patients with primary pulmonary valvular stenosis in whom the infundibular narrowing will resolve be separated from those in whom it will not prior to surgery. This will permit the evaluation of the need for infundibular resection in addition to valvulotomy. In this paper, evidence is presented indicating that it may be possible to separate these groups by means of quantitative angiocardiographic techniques.

The phenomenon of infundibular hypertrophy secondary to pulmonary valvular stenosis has been noted and reported since Kirklin *et al.*⁷ described 6 such cases in 1953. Brock^{1,2} showed that this infundibular thickening would regress after val-

vulotomy. He discussed 60 patients with high right ventricular pressures post-valvulotomy. Seventeen of the 60 patients had infundibular resections. Of those who did not have infundibular resection, 4 died. All but 8 of the remaining patients showed regression of the infundibular stenosis when examined 2 years postoperatively. Johnson⁶ reported 11 cases, 3 of which had incomplete regression of the infundibular thickening. Engle and his associates⁴ reported 3 patients in whom there were electrocardiographic signs of alleviation of right ventricular hypertrophy 6 to 20 months after valvular surgery. Kjellberg *et al.*⁸ reported a case in which angiocardiography 5 months postoperatively showed relief of the infundibular stenosis.

There is some evidence that the type of surgery performed influences the behavior of the infundibulum. Johnson⁶ showed that infundibular stenosis occurred in 51 per cent of his patients following transventricular valvulotomy and in 77 per cent after open valvulotomy through the pulmonary artery. His explanation was that the latter technique permitted more successful elimination of the valvular stenosis and more complete emptying of the ventricle. The effect on the infundibular stenosis was thus increased.

Efforts to evaluate infundibular stenosis have been directed along several lines. Hosier, Pitts and Taussig⁵ have reported that, if the postoperative right ventricular pressure was below 100 mm. Hg, the heart size decreased and the patients did well. Johnson⁶ stated that the 3 cases with infundibular obstruction which he was unable to relieve also had pressures above 100 mm. Hg.

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Watson *et al.*¹¹ and Parizel¹⁰ have employed angiocardiology to determine the presence and degree of secondary infundibular hypertrophy.

Little *et al.*⁹ measured the base of the pulmonary artery and the infundibulum as seen on angiocardiology to quantitate the degree of infundibular obstruction. They found that the infundibular thickening was significant when the ratio of the diameter of the base of the pulmonary artery to the diameter of the infundibulum in systole was less than 0.45.

MATERIAL AND METHOD

The files of the Medical College of Virginia contained selective angiocardiology of 19 patients with pulmonary valvular stenosis with an intact ventricular septum that were adequate for quantitative study. One patient (N.H.) had associated tricuspid insufficiency and an atrial septal defect with a right-to-left shunt. Another patient (E.W.) had a coarctation of the right pulmonary artery. All others had uncomplicated pulmonary valvular stenosis. Of these 19 patients, 9 had significant infundibular obstruction. Measurements were carried out on these 9 patients.

In addition, 42 cases of tetralogy of Fallot were studied for a comparison of the infundibulum with that in uncomplicated valvular stenosis.

Angiocardiology were made on a biplane Schönander Elema rapid film changer at a focal-film distance of 40 inches. Most films were exposed in a sequence of 6-4-4-4 per second. In every case selective angiocardiology was performed through a side-hole catheter placed in the right ventricle.

Measurements were made of the: (1) infundibulum in systole and diastole; (2) transverse diameter of the chest; (3) transverse diameter of the heart; (4) diameter of the jet of opacified blood passing through the stenotic valve; and (5) diameter of the base of the pulmonary artery.

The measurements were made on the films exposed in lateral projection during

the first second of the sequence.

The infundibulum was measured at its narrowest point in systole and at the same point in diastole. The site of maximal contraction varied from patient to patient. Measurements were made at the level of the location of the crista supraventricularis in some, at the parietal band in others.

RESULTS

Measurements in 42 cases of tetralogy of Fallot revealed that the outflow tract behaves as a fixed stenosis. The lumen is narrowed to approximately $\frac{1}{2}$ the diastolic diameter when the ventricle contracts. The average ratio of the diameter of the infundibulum in systole to diastole was 0.56 with a range of 0.33 to 1.00 (Table I, Fig. 1, *A* and *B*). The shape of the infundibulum in various cases varies from that of a smooth cylinder to those with maximum constriction of the area of the crista supraventricularis with formation of a "third ventricle" (dilated distal right ventricular outflow tract). A few showed anomalous insertions of the septal and parietal bands.

Measurements in the 9 cases of pulmonary valvular stenosis with secondary infundibular hypertrophy are presented in Table II. In these, it was found that the infundibulum was much more dynamic than in tetralogy of Fallot (Fig. 2, *A* and *B*). The average ratio of the diameter of the infundibulum in systole to diastole was 0.25 with a range of 0.11 to 0.30. That is, in the average case the outflow tract in systole was narrowed to $\frac{1}{4}$ its diastolic diameter.

In 4 of the 9 patients, the left pulmonary artery was larger than the right (Table II).

TABLE I

Type	No. of Cases	Average of Systole/Diastole Ratio	Range of Systole/Diastole Ratio
Tetralogy of Fallot	42	.56	.33-1.00
Pulmonary Stenosis	9	.25	.11-.30

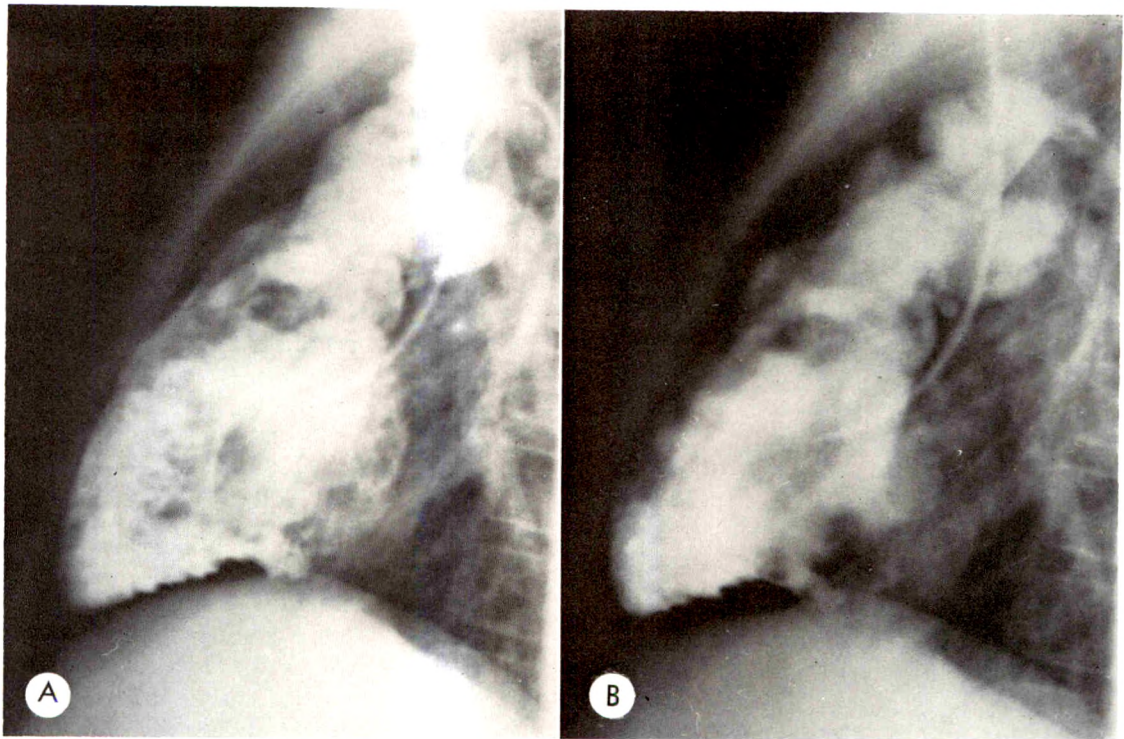


FIG. 1. (A) Tetralogy of Fallot showing infundibular stenosis in diastolic phase. The ventricular septal defect and the aortic opacification through the right to left shunt can also be seen. (B) Same patient in systolic phase. Note the relatively fixed caliber of the infundibular stenosis.

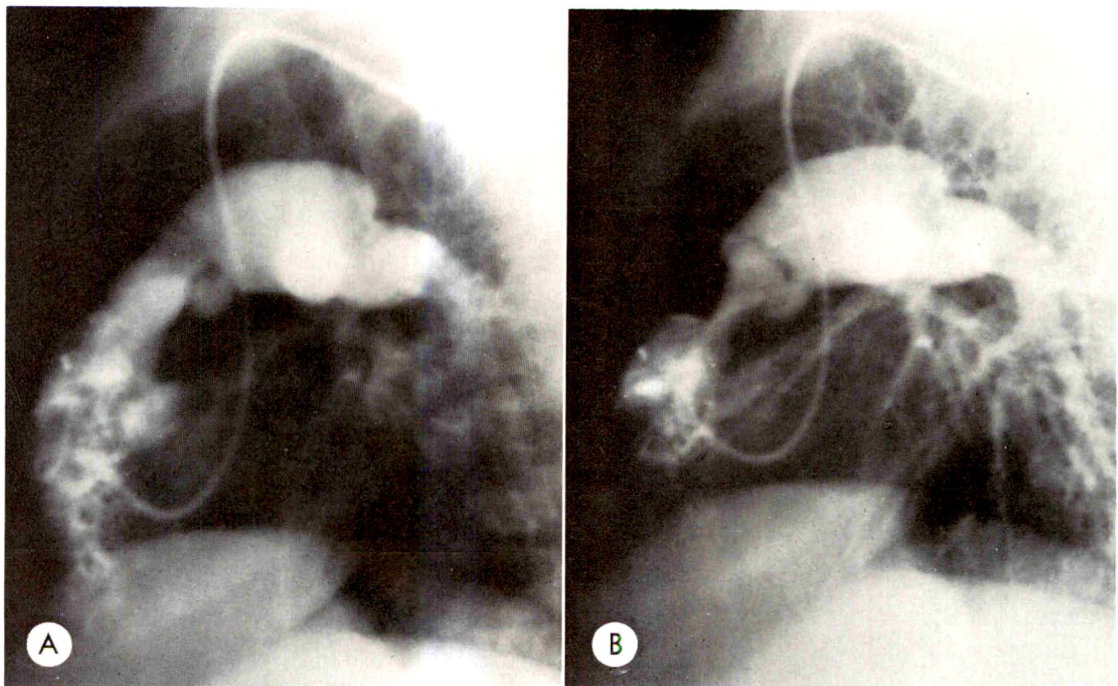


TABLE II

MEASUREMENTS ON ANGIOCARDIOGRAMS OF PATIENTS WITH PULMONARY STENOSIS AND SECONDARY INFUNDIBULAR HYPERTROPHY

Patient	Infundibular Systole (cm.)	Infundibular Diastole (cm.)	Ratio of Systole/Diastole	Transverse Diameter of Chest (cm.)	Transverse Diameter of Heart (cm.)	Diameter of Jet (cm.)	Diameter of Pulmonary Bulb (cm.)	Ratio of Jet/Bulb	Ratio of Systole/Bulb	Diameter 1 cm. Distal to Bifurcation		Other Anomalies
										LPA (cm.)	RPA (cm.)	
J.D.	.6	2.0	.30	20.0	10.0	.6	1.2	.5	(Little) .50	1.1	1.1	—
N.H.	.3	1.0	.30	16.0	9.5	.2	.9	.2	.30	.8	.7	TI ASD
R.D.	.3	1.2	.25	19.0	10.0	.9	1.8	.5	.60	1.6	1.6	—
G.A.	.2	1.2	.17	21.0	12.0	.7	1.2	.6	.17	1.6	1.3	TI ASD R-L Shunt
D.B.	.4	1.6	.25	19.0	10.3	.7	1.6	.4	.25	1.3	1.0	—
D.E.	.5	1.8	.28	20.5	11.7	.9	2.0	.45	.25	2.0	1.8	—
M.S.	.45	1.5	.30	22.5	12.0	.9	1.8	.50	.25	1.6	1.6	—
L.W.	.8	2.7	.11	29.0	17.0	1.8	2.8	.64	.11	3.0	1.5	—
E.W.	.8	2.8	.29	24.0	13.7	1.1	2.6	.42	.31	2.1	2.1	Coarctation of RPA

LPA=Left pulmonary artery.

RPA=Right pulmonary artery.

Jet=Opacified blood passing through the stenotic valve.

TI=Tricuspid insufficiency.

ASD=Atrial septal defect.

Catheterization findings are presented in Table III. Of the 8 patients in whom data were available, only 4 had pressure gradients from the inflow to the outflow tract of the right ventricle.

Five of the patients were subjected to surgery. Following open valvulotomy under hypothermia, 1 patient (M.S.) had a right ventricular pressure of 75 mm. Hg. One year later his right ventricular pressure had decreased to 45 mm. Hg, indicating regression of the infundibular stenosis. Another patient (L.W.) had an extremely active infundibulum which was 9 times as wide in diastole as it was in systole (Fig. 3, *A* and *B*). Because the outflow tract was constricted to 2 mm. in systole, it was felt that valvulotomy without infundibular resection

might result in occlusion of the outflow tract by the hypertrophied muscles. Therefore, valvulotomy and infundibular resection were carried out.

DISCUSSION

At least 3 factors may prevent adequate function of the right ventricle during the period immediately following valvulotomy for pulmonary valvular stenosis: (1) the pulmonary annulus may be too small; (2) the right ventricular cavity may be inadequate; and (3) there may be obstruction of the outflow tract. In the patients considered here, neither the annulus nor the ventricular cavity appeared small. The study was, therefore, concentrated on the third factor; namely, the infundibular stenosis.



FIG. 2. Pulmonary valvular stenosis with secondary infundibular hypertrophy. (*A*) Diastole. (*B*) Systole. The dynamic nature of the infundibular thickening is well demonstrated. The ratio is 0.25 in this case. Note also the doming of the pulmonic valve in systole and the jet of opacified blood across the valve.

TABLE III
CATHETERIZATION PRESSURES AND CLINICAL DATA

Patient	Age	Inflow RV	Outflow RV	RA	MPA	Cyanosis	Heart Murmur	Surgery
J.D.		$\frac{85-90}{5}$	$\frac{75-80}{5}$	—	$\frac{20-25}{5-10}$	None	Grade III at LICS radiating along carotids	None
N.H.		Not available				None	—	—
R.D.	6	$\frac{110-120}{5-10}$	$\frac{110-120}{5-10}$	—	$\frac{25}{15}$	None	Grade III at 3 LICS P ₂ split	—
G.A.	1	$\frac{105-115}{5-10}$	$\frac{105-115}{5-10}$	10	—	None	Grade IV at 2-3 LICS	—
D.B.	27 mo.	$\frac{150}{0}$	$\frac{150}{0}$	—	—	Circumoral	Grades IV over entire precordium	Valvulotomy
D.T.	5	$\frac{105}{5-10}$	$\frac{105}{5-10}$	—	$\frac{2}{10}$	None	Grade IV in pulmonic area	Valvulotomy
M.S.	5	$\frac{75}{5}$	$\frac{45}{5}$	—	$\frac{25}{5}$	Circumoral	Grade IV at 2-3 LICS	Valvulotomy under hypothermia
L.W.	40	$\frac{125}{10}$	$\frac{100-105}{10}$	—	$\frac{25}{5}$	Cyanosis on exertion	Grade III at 3-4 LICS	Valvulotomy and infundibular resection
E.W.	8	$\frac{75}{0}$	$\frac{45}{0}$	—	$\frac{25}{5-10}$	Circumoral	Grade IV at 3 LICS	Valvulotomy

RV=Right ventricle.
RA=Right atrium.

MPA=Main pulmonary artery.
LICS=Left intercostal space.

Eight of the 9 patients in this study met the criteria proposed by Little *et al.*⁹ to determine the significance of secondary infundibular hypertrophy, *i.e.*, infundibulum to pulmonary bulb ratio less than 0.45 (Table II).

In evaluating patients with a combined valvular and infundibular stenosis, 2 factors must be considered. First, the effect of the infundibular obstruction may be enhanced by relieving the valvular stenosis, resulting in heart failure during the postoperative period. Secondly, the hypertro-

phied outflow tract may not regress after valvulotomy.

Patients with a moderate degree of infundibular stenosis tolerate surgery well. For example, 1 patient (D.B.) (Fig. 2, *A* and *B*) had a systolic/diastolic infundibular ratio of 0.25 and a systolic/bulb ratio of the same. Even with this degree of stenosis, she had a successful postoperative course. On the other hand, in patient L.W. (Fig. 3, *A* and *B*) infundibular resection in addition to pulmonary valvulotomy was carried out because the marked narrowing of the infun-

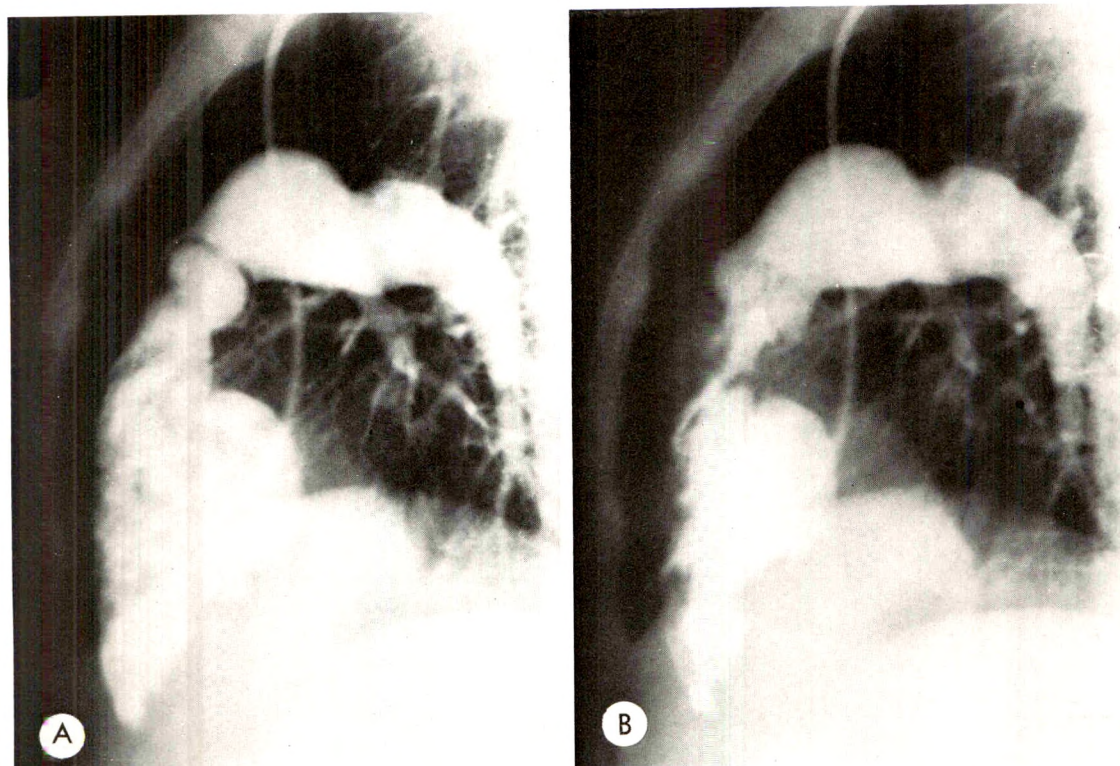


FIG. 3. Pulmonary valvular stenosis with secondary infundibular hypertrophy. (A) Early systole before maximal contraction of the infundibulum. The domed valve and the jet are demonstrated. (B) Late systole. The valve has closed. The systolic-diastolic ratio is 0.11.

dibulum in systole suggested that the infundibular stenosis might be enhanced by relieving the valvular stenosis.

Of the 4 patients who did not have infundibular resections, 1 had catheterization proof of regression and the others had electrocardiographic changes in the direction of normal. Therefore, none of the patients studied appeared to have nonregressing infundibula.

A comparison of the data in these 9 patients with those obtained from the 42 patients with tetralogy of Fallot provides a basis for conjecture about the nonregressing infundibulum. We have shown that in these conditions there is a significant difference in the movement of the infundibular muscles during the cardiac cycle. It would seem that any change in the hypertrophied infundibulum of pulmonary valvular stenosis that would make it more like the fixed stenosis of tetralogy of Fallot would decrease

the chance of regression after valvulotomy. There is some indication that the amount of fibrous tissue in the infundibulum of patients with pulmonary valvular stenosis increases with age.³ It seems likely that this is related to an increased potential for failure to regress with age.

The following points are emphasized: (1) the significance of the infundibular obstruction may be determined by the Little *et al.*⁹ ratio of systolic diameter of the infundibulum/diameter of the pulmonary bulb; (2) in all cases of tetralogy of Fallot, the measurements made in this study (infundibular systolic diameter/infundibular diastolic diameter) were greater than 0.33; (3) in all cases of pulmonary valvular stenosis in this series, the infundibular systolic/diastolic ratio was less than 0.30; and (4) on this basis, one may predict that an infundibulum with significant obstruction as measured by Little and co-workers' ratio and an

infundibular systolic/diastolic ratio greater than 0.30 may not regress following valvulotomy.

The incidental finding that the left pulmonary artery was larger than the right in 4 of our patients was the result of jet streams of blood through the stenotic valve directed toward the left pulmonary artery. A reflection of this is often seen during fluoroscopic studies where the left pulmonary artery pulsates more vigorously than the right.

SUMMARY

The behavior of the infundibulum in 9 cases of pulmonary valvular stenosis with secondary infundibular thickening has been compared with 42 cases of tetralogy of Fallot. Data are presented to show that the hypertrophied infundibulum expands and contracts to a much greater degree than the infundibulum in tetralogy of Fallot.

A quantitative technique is presented for distinguishing the dynamic from the fixed infundibular stenosis in patients with pulmonary valvular stenosis and secondary infundibular thickening. It is believed that the ratio employed by Little *et al.*⁹ (systolic diameter of the infundibulum/diameter of the pulmonary bulb) combined with the infundibular systolic/diastolic ratio may provide objective evidence for a fixed infundibular stenosis that will require resection in addition to pulmonary valvulotomy.

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FUNCTIONAL PULMONARY ATRESIA*

A CAUSE OF ANGIOCARDIOGRAPHIC MISINTERPRETATION IN TETRALOGY OF FALLOT

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TETRALOGY of Fallot is the most common cyanotic congenital cardiac defect which is compatible with life beyond infancy and early childhood.¹³ For the preoperative evaluation of this defect, the angiocardio-graphic examination should allow an assessment of the condition of the outflow tract of the right ventricle, with special reference to the character of the infundibular stenosis.¹¹ In 1954, Hilario *et al.*¹⁰ noted that there is often a discrepancy between the appearance of the right ventricular outflow tract on angiocardio-graphy and the results of catheterization across it. They used serialographic intravenous angiocardio-graphs to demonstrate that the form and caliber of the infundibulum of the right ventricle in Fallot's tetrad has great variations during different phases of the cardiac cycle. The contraction of the infundibulum was occasionally sufficient to occlude almost completely the infundibulum during systole. These observations were verified hemodynamically by Rodbard and Shaffer¹⁵ by the simultaneous recording of the pressures in the right ventricular cavity and in the infundibulum. It was demonstrated that in tetralogy of Fallot the pressures in the two chambers began to rise together during isometric contraction and for a variable part of the ejection phase (usually 0.1 sec.), but that the infundibular pressure then began to fall, even though the right ventricular pressure rose. It was postulated that this was due to dynamic narrowing of the infundibulum during early or mid-systole, probably due to muscular contraction.

It was once thought that the obstruction

to pulmonary blood flow in tetralogy of Fallot was fixed.⁸ Catheterization studies, however, have subsequently demonstrated that, in addition to the cyclical alterations of the cross-sectional area of the right ventricular infundibulum, persistent spasm of the infundibulum could occur.^{1,2,3,4,17} The magnitude of the infundibular obstruction was shown to become more severe coincident with the appearance of or increase in cyanosis, or the loss of consciousness. A consistent shortening, softening and eventual disappearance of the pulmonary systolic murmur was noted, paralleling the fall in arterial oxygen saturation. When unconsciousness occurred, no murmur at all was heard, suggesting a state of functional pulmonary atresia.¹⁷ The purpose of this communication is to report and discuss the phenomenon by which the angiocardio-graph in a patient with tetralogy of Fallot may present the appearance of pulmonary atresia (Fig. 2, A-H) when, in fact, the right ventricular outflow tract is patent.

REPORT OF A CASE

P.G., N.I.H. #02-16-49. This 6 year old white female was admitted to the National Heart Institute on September 28, 1958. A heart murmur and cyanosis had been present since birth. While her growth and development were normal, she was dyspneic with exertion and squatted to rest. She had episodes of intense paleness and weakness without syncope which lasted 5 to 10 minutes, followed by tachycardia and intense cyanosis. Conventional roentgenograms revealed a "coeur en sabot" configuration. At cardiac catheterization in June, 1954, right ventricular pressure was 80/0 and femoral artery pressure was 85/70. The pulmonary ar-

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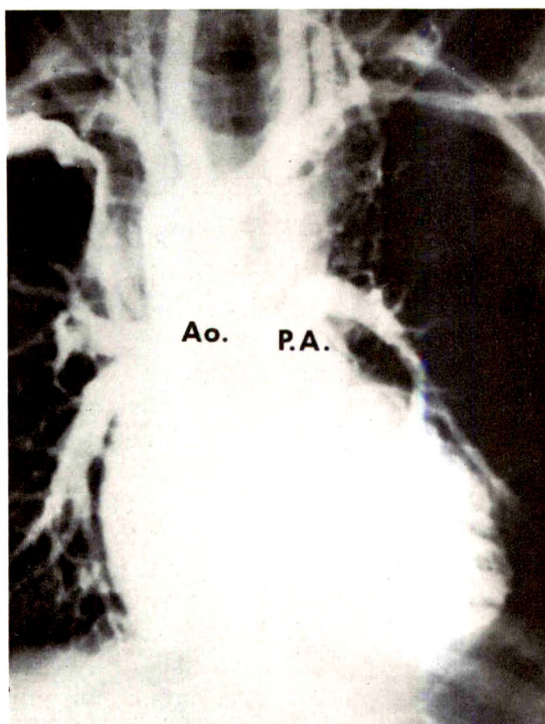


FIG. 1. Intravenous angiogram made in 1955 demonstrating excellent simultaneous filling of the aorta and pulmonary arteries. Ao. = aorta; P.A. = pulmonary artery. (Courtesy of N. A. Antonius, M.D., St. Michael Hospital, Cardiac Center, Newark, New Jersey.)

tery was not entered. A peripheral venous angiogram at an outside hospital (Fig. 1) showed simultaneous filling of the aorta and pulmonary artery, and a right aortic arch. Physical examination on admission in 1958 revealed a deeply cyanotic girl with a blood pressure of 94/70 and a regular pulse of 88. The pulmonic second sound was present but was diminished. There was a harsh systolic murmur along the left sternal border, loudest in the left second intercostal space. On this admission, right heart catheterization revealed a right ventricular pressure of 100/0 and a pulmonary artery pressure of 9/4. A nitrous oxide test in the right ventricle was 27 per cent while a right ventricular cardiogreen dye curve showed a right-to-left and a left-to-right shunt. Right ventricular angiography (Fig. 2, A-H) demonstrated a high interventricular septal defect with filling of the left ventricle. All of the contrast material from the right ventricle appeared to go out the aorta. A narrowed, pointed infundibulum of the right ventricle ended

abruptly and had the appearance of an atretic outflow tract. Because of the misleading angiographic findings, a subclavian-pulmonary artery anastomosis was elected as the treatment of choice and was performed. Exploration at the time of surgery demonstrated that the outflow tract of the right ventricle was patent and morphologically amenable to complete correction and that the pulmonary arteries were adequately developed.

Postoperatively, the patient did well and regression of the right ventricular hypertrophy was noted on the electrocardiogram. Four years later, however, she developed increased cyanosis, lack of energy and chest pain on mild exertion. No signs of congestive failure were present. She was readmitted on July 21, 1963 at which time a harsh Grade III/VI systolic murmur was heard along the left sternal border. Although a soft continuous murmur was heard over the left chest, it was thought that the systemic-pulmonary artery anastomosis was no longer adequate and that a Brock-type pulmonary valvotomy and infundibular resection was indicated. At operation, the interventricular septal defect was noted to be high in the membranous septum and to measure about 2 cm. in diameter. Above this level a discrete concentric "bar-like" narrowing of the outflow tract was found which was continuous with the crista supraventricularis. The pressure in the right ventricle was 75 mm. Hg and equal to systemic. A gradient of about 25 mm. Hg appeared to be present across the pulmonic valve and a gradient of 25 to 30 mm. Hg was present across the discrete infundibular obstruction, about 2 cm. proximal to the pulmonic valve. After pulmonary valvotomy and infundibular resection, the pulmonary artery pressure was 15 mm. Hg and the right ventricular pressure was between 55 and 60 mm. Hg. During the procedure, the patient developed a right bundle branch block.

In the 8 months following this second surgical procedure, the patient had several episodes of congestive failure which ultimately became intractable. She was thus readmitted on February 12, 1964 at which time she had a Grade III/VI pansystolic murmur and a short diastolic murmur along the left sternal border. After appropriate therapy which improved her condition moderately, a cardiac catheterization revealed a pulmonary artery pressure of 90/25 and a radioactive krypton of 83 per cent. The right ventricular pressure was also 90/25 with a

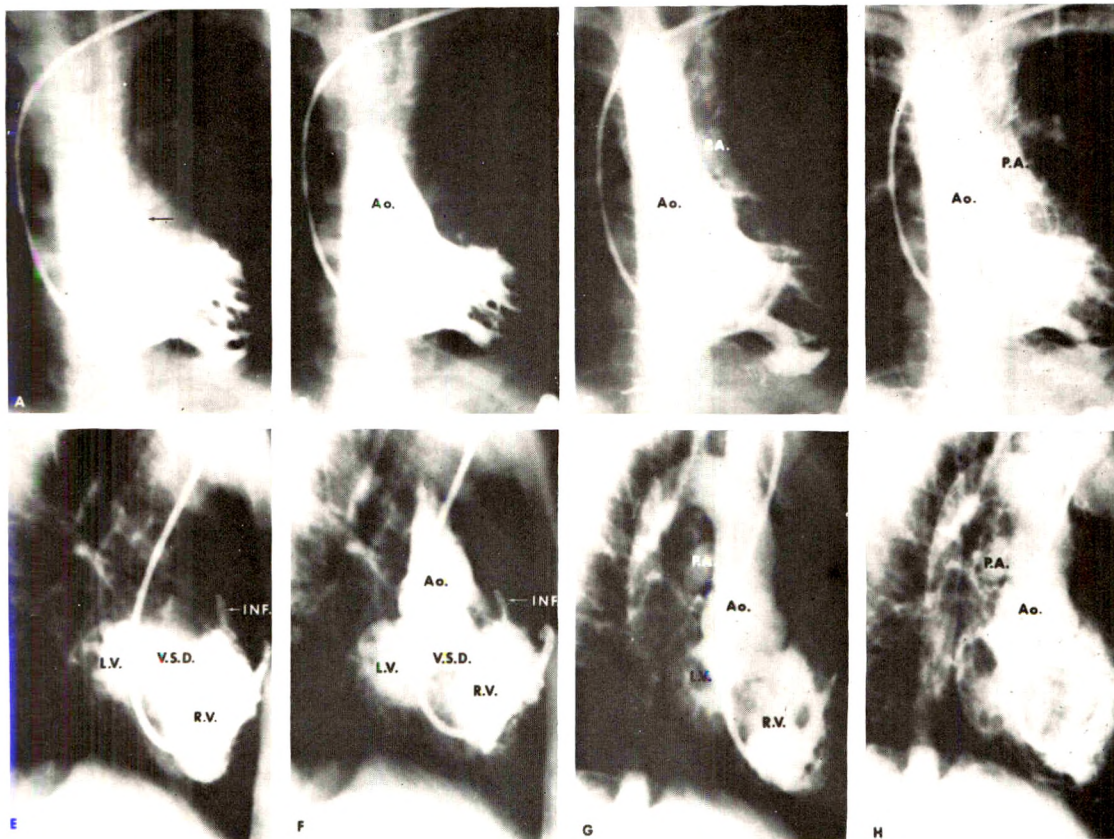


Fig. 2. Selective right ventricular angiograms of the same patient as in Figure 1, made in 1958 in frontal (A through D) and lateral (E through H) projections, demonstrating an appearance of pulmonary atresia in a patient with tetralogy of Fallot who had patency of the pulmonary outflow tract subsequently proved. This series of roentgenograms demonstrates a small amount of contrast material in the right ventricular infundibulum (INF.) in E and F. The tapered contrast shadow in A (arrow) probably represents contrast material beneath the aortic valve, although it might be interpreted as being in the right ventricular infundibulum. The only great vessel which can be seen filling from the ventricles is the aorta (Ao.). The main pulmonary artery (P.A.) is seen to opacify faintly, but the source of the opacification is not identified. The left and right ventricular cavities (L.V. and R.V.) and the ventricular septal defect (V.S.D.) are clearly delineated. Note that a portion of the contrast material was injected into the myocardium anteriorly (E and F).

radioactive krypton of 77 per cent. The right atrial mean pressure was 20 mm. Hg. The impression was that of a left-to-right shunt through the ventricular septal defect with a pulmonary-to-systemic flow ratio of 6:1, pulmonary hypertension, severe pulmonary insufficiency and tricuspid insufficiency. It was felt that the patient was in the best clinical condition which could be achieved, and she was taken to the operating room with the idea of closing the ventricular septal defect. However, cardiac arrest occurred upon induction of anesthesia. A cardiopulmonary bypass was instituted and the septal defect was closed, but cardiac action was inadequate after termination of perfusion and

resuscitation was not possible. Postmortem examination revealed the findings of tetralogy of Fallot in which a Blalock shunt and a Brock infundibular resection had been performed (Fig. 3).

DISCUSSION

Although the obstruction to pulmonary blood flow in this patient was very severe, the presence of a harsh pulmonic systolic ejection murmur, the passage of a catheter into the pulmonary artery during right heart catheterization, the demonstration of excellent filling of the pulmonary arteries

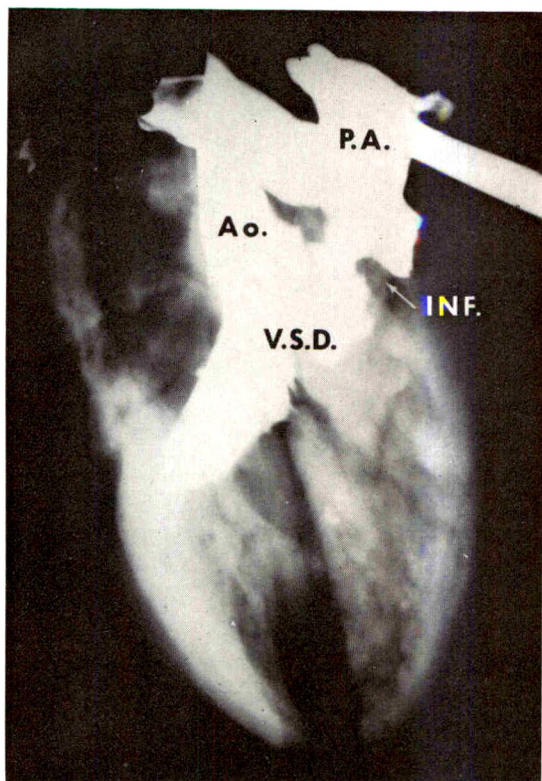


FIG. 3. Postmortem roentgenogram of barium injected heart of patient with tetralogy of Fallot whose angiocardiograms are shown in Figures 1 and 2, demonstrating that the infundibulum of the right ventricle (INF.) is not atretic. The pulmonary artery (P.A.), aortic root (Ao.) and ventricular septal defect (V.S.D.) are seen filled with contrast material.

simultaneously with the aorta on an earlier occasion (Fig. 1), and the demonstration of a developed outflow tract at surgery all served to verify that this patient did not have pulmonary atresia. The possible explanations for diminished filling of the pulmonary arteries during angiocardiography in tetralogy of Fallot, in addition to infundibular spasm, include marked peripheral vasodilatation causing a large amount of right-to-left shunting, and pulmonary vasoconstriction causing a marked increase in pulmonary vascular resistance. It has been shown by Wood¹⁷ and by Braudo and Zion,² however, that in tetralogy of Fallot, neither an increase in pulmonary vascular resistance nor a decrease in peripheral resistance need occur during

episodes of increased right-to-left shunting such as is seen with cyanotic spells or syncope. The role of spasm of the right ventricular outflow tract, occurring spontaneously or due to cardiac catheterization, on the other hand, has been implicated in the production of increased cyanosis and syncopal spells in such patients.^{1,2,3,4,17} These views have been recently challenged by Guntheroth and co-workers⁷ who believe that hyperpnea can lead to an increased systemic return with a fixed or decreased pulmonary blood flow, causing an increase in right-to-left shunting. This in turn leads to an increased arterial $p\text{CO}_2$ and a decreased $p\text{O}_2$ and pH, which leads to further hyperpnea. He further challenges infundibular spasm as the sole cause for cyanotic spells in tetralogy of Fallot since patients with true pulmonary atresia also have cyanotic spells, as was also pointed out by Wood.¹⁷ However, evidence for the occurrence of critical functional narrowing of the right ventricular outflow tract in tetralogy of Fallot was presented recently by Cumming⁵ as the result of a study of the effects of isoproterenol. He postulated that the anoxic spells may be precipitated by peripheral vasodilatation and/or increased circulating catecholamines. Similar evidence indicating that muscular obstruction to right ventricular outflow is dynamic and that it may vary in severity in patients was presented by Mason and co-workers,¹² while similar findings in dogs were presented by Harwood and co-workers.⁹

Another possible mechanism for an erroneous appearance of the right ventricular outflow tract on angiocardiograms is the occurrence of an arrhythmia at the time of angiocardiography, which does not allow the infundibulum to relax. In the patient presented, ventricular premature contractions did occur during angiocardiography and may have contributed to the failure of the pulmonary arteries to fill from the right ventricle.

Whatever the mechanism by which the phenomenon occurred, a persistently nar-

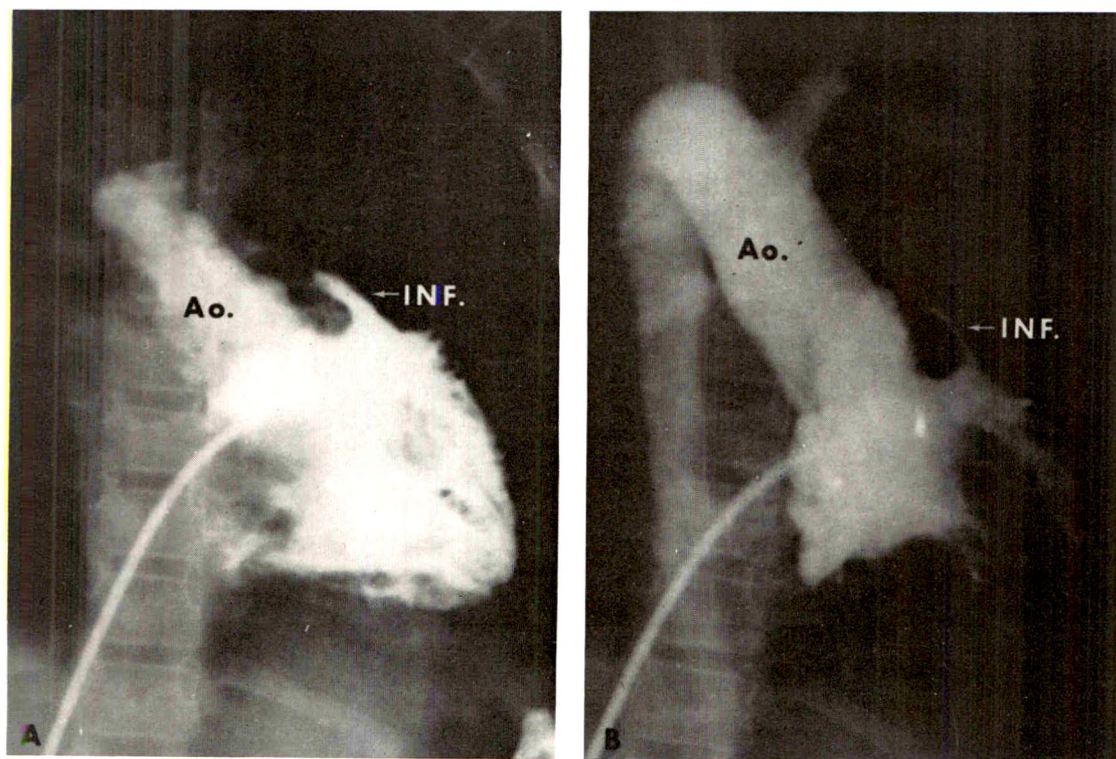


FIG. 4. Frontal angiocardigrams in diastole (*A*) and systole (*B*) of a patient with proved pulmonary atresia demonstrating the change in caliber of the right ventricular infundibulum (INF.) during the cardiac cycle. Note incidentally the right sided aortic arch (Ao.).

rowed and tapered appearing right ventricular infundibulum was noted (Fig. 2, *A-H*) which suggested that this patient had pulmonary atresia. The angiocardigrams of 2 patients with surgically verified pulmonary atresia are shown in Figures 4, *A* and *B*; and 5, *A* and *B*. Note that there are cyclical alterations in the right ventricular infundibulum in these 2 patients, while this is not noted in the angiocardigrams of the patient with functional pulmonary atresia (Fig. 2, *A-H*).

From the case reported, it is apparent that in patients with tetralogy of Fallot in whom the pulmonary artery cannot be entered during catheterization, it is possible to make the misdiagnosis of pulmonary atresia from the angiocardigram when, in fact, the pulmonary outflow tract is patent and may even be adequate for complete correction. The prognostic significance of such an error is that the patient

might be treated with a shunt instead of a more physiologic infundibular resection or a total correction.

In order to avoid the pitfall of incorrectly diagnosing pulmonary atresia in patients with tetralogy of Fallot who have functional pulmonary atresia at the time of angiocardigraphy, the following rule of thumb is suggested. If no contrast material is seen entering the pulmonary artery from the right ventricle (Fig. 2, *A-H*; 4, *A* and *B*; and 5, *A* and *B*), and if there are cyclical contractions of the infundibulum of the right ventricle (Fig. 4, *A* and *B*; and 5, *A* and *B*), then pulmonary atresia may be diagnosed with some assurance. If, however, the atretic-appearing infundibulum is not seen to alter its dimensions during the study (Fig. 2, *A-H*), the possibility of functional pulmonary atresia should be considered.

After a systemic-pulmonary artery anas-

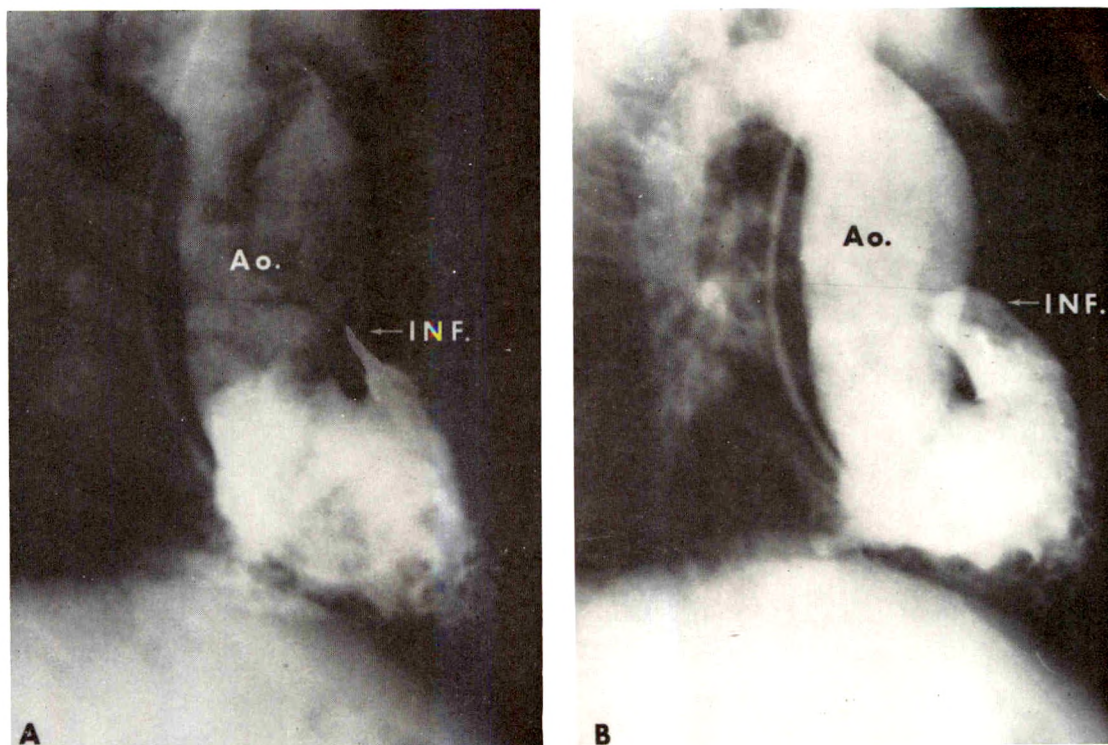


FIG. 5. Lateral angiocardigrams of another patient with proved pulmonary atresia demonstrating the marked change in caliber of the right ventricular infundibulum (INF.) between systole (A) and diastole (B).

tomosis, a tetralogy of Fallot's right ventricular obstruction may be increased to the point of being converted into pulmonary atresia.^{6,14,16} This does not apply to the reported case, however, since both angiocardigraphies were performed before the systemic-pulmonary artery shunt was made.

An item of further interest regarding this patient is the development of pulmonary hypertension after infundibular resection and pulmonary valvotomy. It has been postulated by Brock³ that the probable role of the infundibulum is to support the pulmonic valve so that, with increasing degrees of pulmonary artery pressure, the hypertrophy of the right ventricle which occurs simultaneously increases the support of the valve by way of the infundibulum, thus preventing pulmonary regurgitation. In fact, Hilario *et al.*¹⁰ thought that infundibular contraction may explain why pulmonary valvotomy in tetralogy of Fallot does not

transform this condition into an Eisenmenger's complex, the relatively moderate degree of infundibular stenosis acting as a protective mechanism by regulating transmission of the ventricular pressure to the pulmonary vessels. In the patient herein reported, this protective mechanism was apparently lost during the combined pulmonary valvotomy and infundibular resection so that due to the operation, the pulmonary artery pressure rose from 10 mm. Hg systolic to 90 mm. Hg systolic. Thus, the condition in this patient had been transformed into a ventricular septal defect with pulmonary hypertension, followed eventually by ventricular failure.

SUMMARY AND CONCLUSIONS

A patient with tetralogy of Fallot is reported in whom angiocardigraphic examination revealed the findings of pulmonary atresia, while clinical, catheterization and operative findings indicated that the pulmonary outflow tract was

patent. The possible mechanisms for the atretic appearance of the outflow tract of the right ventricle and the failure of the pulmonary arteries to fill with contrast material from the right ventricle are discussed. Since the appearance of the right ventricular infundibulum during angiocardiology can lead to an erroneous impression of the condition of the infundibulum, it is important that the phenomenon of functional pulmonary atresia on angiocardigrams of patients with tetralogy of Fallot be recognized. It is suggested that functional pulmonary atresia may be differentiated angiocardigraphically from true pulmonary atresia by observing cyclical alterations of the infundibulum in the latter.

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TETRALOGY OF FALLOT*

A DETAILED ANGIOCARDIOGRAPHIC STUDY

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and ROBERT T. OSTEEN, A.B.†

TETRALOGY of Fallot is the most common cardiac malformation associated with cyanosis. It accounts for 15 per cent of all congenital heart disease¹² and 50–75 per cent of all cyanotic heart disease beyond 2 years of age.⁷ Fallot's anatomic syndrome consisted of pulmonary stenosis or atresia, dextroposition of the aorta, interventricular septal defect, and right ventricular hypertrophy.¹⁵

The clinical features include retarded physical development, cyanosis, and dyspnea leading to anoxic episodes and squatting. Pertinent physical findings usually include an undisplaced apical beat, a loud aortic second sound, and a precordial heave. The characteristic murmur is short, harsh and systolic. It is located in the third and fourth interspace at the left sternal border with an associated thrill. Signs of congestive failure are not usually present in childhood.⁷

The conventional roentgenographic examination reveals slight to moderate cardiomegaly and an enlarged right ventricle usually characterized by an upturned apex and anterior bulging. The left cardiac border is most often concave at the site of the pulmonary artery. Characteristically, there is sparse pulmonary arterial vascularization in the hilar regions and in the lungs in patients with clinical cyanosis or significant desaturation. Left atrial enlargement is absent and the aorta is usually dilated. A right aortic arch is reported to be present in slightly more than 20 per cent of all cases.^{5,8}

The electrocardiogram is characterized by right ventricular hypertrophy.

Selective right ventricular angiocardiography and cardiac catheterization have been improved upon considerably in recent

years so that we can more accurately appraise the anatomic and physiologic changes in congenital heart disease. In this study we plan to show the value of selective angiocardiography in estimating and predicting the future course and management of individuals with tetralogy of Fallot.

METHOD AND MATERIAL

The study includes 47 patients with tetralogy of Fallot who underwent selective angiocardiography at the Medical College of Virginia Hospital between 1959 and 1963. One exception is a well demarcated venous angiogram made in 1954. Variants of pulmonary atresia with ventricular septal defect ("pseudotruncus arteriosus") and origin of both great vessels from the right ventricle with pulmonary stenosis, are excluded.

In 46 cases the catheter was placed in the right ventricle. In 2 of these cases the catheter recoiled from the right ventricle to the right atrium during the injection.

The technique consisted of high pressure injection of contrast material through a side-holed catheter. This was accompanied by rapid, serial, biplane roentgenography at a 40 inch focal-film distance utilizing the Schönander apparatus.

Numerous measurements and observations were recorded, utilizing anteroposterior and lateral projections at different phases of the cardiac cycle (Fig. 1, *A* and *B*; and 2, *A* and *B*). Parameters evaluated included the diameters of the chest, heart, main pulmonary artery, ascending aorta, and ventricular septal defect uncorrected for dilatation. The infundibulum of the right ventricle was measured at its narrowest point in maximum contraction and

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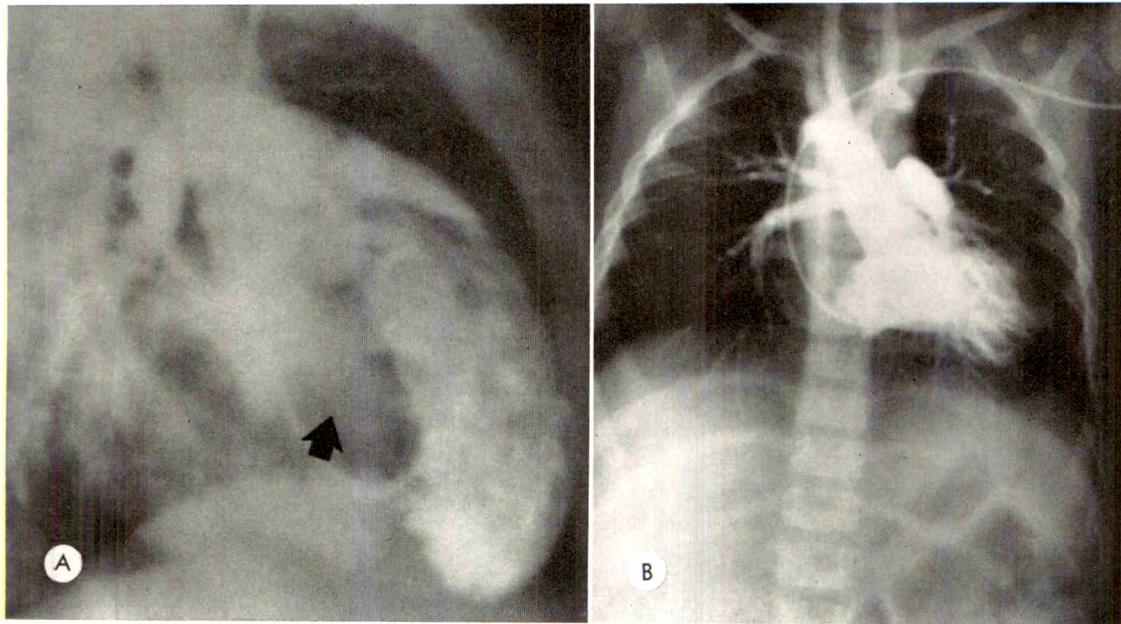


FIG. 1. Selective angiocardiograms from right ventricle, 5 year old boy. (A) Lateral view. Arrow points to ventricular septal defect. There is only minimal "overriding." (B) Frontal view. There is a right aortic arch.

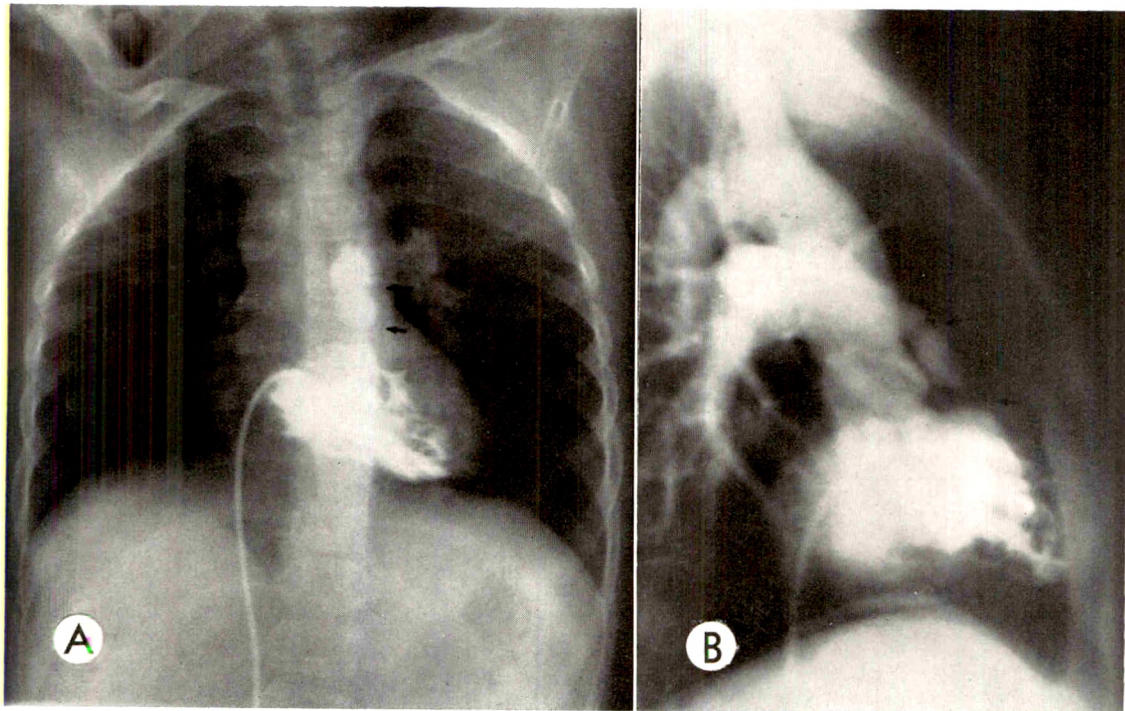


FIG. 2. Selective angiocardiograms from right ventricle, 8 year old girl. (A) Frontal view, early study. Lower arrow points to area of infundibular stenosis, upper arrow to valvular stenosis. (B) Slightly later study in lateral view. The left ventricle and aorta are now filled through a large ventricular septal defect. Upper and lower arrows as in A.

TABLE I
ANGIOCARDIOGRAPHIC DATA
(45 cases)

Measurement	Average	Median	Range
Infundibular Stenosis			
Systole: Anteroposterior Diameter (cm.)	0.46	0.30	0.1-1.2
Lateral Diameter (cm.)	0.40	0.40	0.1-1.2
Transverse Diameter of Heart/Systolic Stenosis	38.2	31.7	10.1-150.0
Diastole: Anteroposterior Diameter (cm.)	0.86	0.80	0.2-2.4
Lateral Diameter (cm.)	0.88	0.80	0.3-2.1
Transverse Diameter of Heart/Diastolic Stenosis	15.0	17.7	4.6-53.5
Main Pulmonary Artery Diameter (cm.)	1.7	1.6	0.6-4.0
Transverse Diameter of Chest/Main Pulmonary Artery	13.9	13.3	5.1-25.8
Aorta Diameter (cm.)	2.5	2.2	1.2-4.3
Aorta Diameter/Transverse Diameter of Chest	0.114	0.112	.068-.174
Interventricular Septal Defect Diameter (cm.)	1.3	1.1	0.4-4.0

maximum dilatation (Table I and II).

Observations were made as to the relative opacity of the main pulmonary artery to the aorta, shape of the infundibular chamber, and direction of the aortic arch.

The degree of overriding of the aorta on the ventricular septum and the direction of tilt of the aortic valve plane were assessed. The presence of other cardiovascular anomalies was also recorded.

TABLE II
ANGIOCARDIOGRAPHIC DATA

Factor	Total No. of Cases	No. Affected	Per Cent
Sex	47		
Males		27	57
Females		20	43
Right Aortic Arch	50	4	8
Pulmonary Valvular Stenosis	47	17	35
Shape of Infundibulum	47		
Cone		22	47
Cylinder		25	53
Overriding of the Aorta	44		
0-25%		31	70
25-50%		3	7
50-75%		8	18
75-less than 100%		2	4
Aortic Valve Plane Direction	45		
Transverse		5	11
Anterior		22	49
Posterior		18	40
Density of Aorta to Main Pulmonary Artery	47		
Aorta = Main Pulmonary Artery		11	23
Aorta > Main Pulmonary Artery		6	13
Main Pulmonary Artery > Aorta		30	64

The clinical records on all patients were reviewed with regard to history and physical findings (Table III). Cardiac catheterization data (Table IV) and pathologic findings at surgery or postmortem examination were recorded when available.

Computer techniques were employed in correlating angiocardigraphic findings with clinical observations and predicting the clinical course of future patients by the observations at angiocardiology. The technique involved a multiple linear regression on a RPC-4000 computer. The coefficients of determination were calculated by the method of least squares. Student's *T* values were used for levels of significance at 95 per cent confidence with *N-P-1* degrees of freedom.

FINDINGS

1. *Age and Sex.* The age at the time of angiocardiology ranged from 2 months to 32 years with an average of 6.7 years and a median of 5.3 years. There were 27 males (57 per cent) and 20 females (43 per cent). This sex distribution is consistent with the 57 per cent males reported by Fontana and Edwards⁵ and the 59 per cent males reported by Keith, Rowe, and Vlad.⁷

2. *Infundibular Stenosis.* A nearly equal incidence of configuration of the infundibulum was found. The infundibular chamber was cylinder shaped in 53 per cent and cone shaped in 47 per cent.

The infundibular stenosis in tetralogy of Fallot is relatively fixed, changing only two-fold between systole and diastole as compared to four-fold in pulmonic stenosis with an intact ventricular septum.⁹ This demonstrates that the secondarily hypertrophied infundibulum expands and con-

TABLE III
CLINICAL DATA

Factor	Total No. of Cases	No. Affected	Per Cent
I. Growth	47		
decreased		30	64
normal		17	36
II. Dyspnea on Exertion	47		
absent		13	28
present		34	72
mild		21	44
severe		13	28
III. Cyanosis	47		
absent		14	30
present		33	70
mild		11	23
severe		22	47
IV. Clubbing	47		
absent		23	49
present		24	51
mild		17	36
severe		7	15
V. Squatting (over 2 yr.)	39		
absent		15	38
present		24	62
mild		18	46
severe		6	16
VI. Polycythemia	47		
greater than 15 gm. per cent		26	62
less than 15 gm. per cent		21	38

tracts to a greater degree than the infundibulum in tetralogy of Fallot.

Pulmonary valvular stenosis was found in 17 cases (35 per cent), characterized by doming or thickening of the valve leaflets, poststenotic dilatation, or a narrowed orifice with a jet phenomenon. Other authors describe valvular stenosis in 36 to 82 per cent of their cases.^{5, 7, 8, 12.}

TABLE IV
CATHETERIZATION DATA

Measurement	Total	Average	Median	Range
Right Ventricular Pressure (mm.)	41	93	90	65-135
Right Ventricular Oxygen Saturation (%)	31	71	71	45-94
Right Atrial Oxygen Saturation (%)	24	64	66	43-80

TABLE V
RIGHT AORTIC ARCH IN TETRALOGY OF FALLOT

Investigators	No. Cases Studied	Right Aortic Arch	
		No. Affected	Per Cent
Blalock and Bahnson ²	610	140	23
Dammann <i>et al.</i> ³	108	28	26
Baker <i>et al.</i> ¹	50	14	28
Lowe ¹⁰	50	6	12
Pattinson and Emanuel ¹⁴	60	12	20
Kjellberg <i>et al.</i> ⁸	63	8	13
Fontana and Edwards ⁵	28	5	18
Edwards ⁴	56	11	20
Total	1,025	224	22
Present Study	47	4	8.5
Present Study including Pseudotruncus	50	4	8.0

3. *Overriding of the Aorta.* Keith *et al.*⁷ describe 25 to 50 per cent overriding as being most common. We found 31 (71 per cent) out of 44 patients who were measured exhibiting less than 25 per cent overriding with 22 of these having essentially no overriding. Less than 50 per cent overriding was found in 77 per cent. No patients were included with an aorta totally to the right of the septum since we consider this to be a separate entity, origin of both great vessels from the right ventricle.¹³

The aortic valve plane pointed anteriorly in 49 per cent and posteriorly in 40 per cent.

4. *Right to Left Shunt.* The densities of contrast in the ascending aorta and the main pulmonary artery during the same exposure were compared by direct visualization. Both vessels appeared roughly equal in density in 23 per cent of the patients, with the main pulmonary artery predominating in 64 per cent and the aorta in 13 per cent. The interventricular septal defect was poorly visualized in many cases. Measurements along its longitudinal diameter showed an average of 1.3 cm. and a mean of 1.1 cm.

5. *Right Aortic Arch.* A right aortic arch

was found in only 4 patients or 8.5 per cent. When 3 patients with pulmonary atresia were included, the incidence fell to 8.0 per cent. These figures fall far below the statistics usually reported in the current literature (see Table v).

6. *Clinical Findings.* Twenty patients (43 per cent) were found to have noncardiovascular birth anomalies or a family history of congenital heart disease. Sixty-four per cent revealed evidence of decreased growth, a characteristic of all congenital heart disease with marked cyanosis.⁸ Dyspnea on exertion was present in 72 per cent and cyanosis in 70 per cent. Thirty per cent of all patients were acyanotic. Clubbing was found in 51 per cent. Twenty-four (62 per cent) of 39 patients over 2 years of age had a history of squatting.

7. *Polycythemia.* Hemoglobin concentration levels ranged from 11.4 to 22 gm. per cent with averages of 16.4 gm. per cent in all patients and 17.4 gm. per cent in the 33 cyanotic patients. Sixty-two per cent of all patients and 67 per cent of the cyanotic patients had hemoglobin levels greater than 15.0 gm. per cent. Kjellberg *et al.*⁸ found hemoglobin concentrations of 15 to 26 gm. per cent in 86 per cent of 62 cyanotic cases.

8. *Cardiac Catheterization.* Forty-one right ventricular pressures were recorded ranging from 65–135 mm. Hg, with an average of 93 mm. and a mean of 90 mm. Right ventricular oxygen saturation in 31 patients averaged 71 per cent with 61 per cent saturation in the right atrium.

DISCUSSION

Selected angiocardiographic measurements were computed against pertinent clinical findings to determine significance. Various factors were found to be statistically significant at the 95 per cent level of confidence in predicting the occurrence and severity of clinical components of tetralogy of Fallot (Table VIA and VIB). These significant factors were applied to calculated equations for predicting the occurrence of a clinical finding in future patients. These equations were tested against 44 of the pre-

TABLE VIA
COMPUTED SIGNIFICANCE FOR PREDICTABILITY

Factor	Symbol	Growth y1	Dyspnea on Exertion y2	Squatting y3	Cyanosis y4	Clubbing y5	Hb y6
Transverse Diameter of Chest	×1	.11	.36*	.44*	.20	.12	.33*
Heart/Systolic Stenosis	×2	.11	.56*	.26†	.08	.17*	.24*
Heart/Diastolic Stenosis	×3	.10	.47*	.27	.15	.09	.01
Chest/Main Pulmonary Artery	×4	.04	.19	.24†	.49*	.31*	.38*
Aorta/Chest	×5	.31*	.08	.14†	.14	.10	.10
Aorta/Main Pulmonary Artery Density	×6	.24†	.03	.04	.23	.22†	.07
Overriding of Aorta	×7	.05	.02	.06	.22*	.04	.001

Key: Numbers indicate relative significance as related to other measurements for specific factor only.

* Significant predictor at 95 per cent level.

† Used as predictor in equations.

TABLE VIB
PREDICTABILITY CORRELATION

Factor	Predictor	Correlation (per cent)		
		Over- all	+ or —	1+ or 2+
Decreased Growth	×5, ×6*	70	70	—
Dyspnea on Exertion	×1, ×2, ×3	50	70	60
Squatting	×1, ×4, ×5	60	70	64
Cyanosis	×4, ×7	50	77	62
Clubbing	×2, ×4, ×6	50	60	65
Hemoglobin Concentration within 2 gm. per cent	×1, ×2, ×4	40	—	—
within 3 gm. per cent		66	—	—

* See symbols in Table VIA.

viously measured patients with approximately 70 per cent accuracy in predicting occurrence and approximately 60 per cent accuracy in predicting severity.

The aortic diameter and relative density of aortic to main pulmonary artery contrast were found to be significant in predicting decreased growth. Using these two factors, 70 per cent correlation was found.

For predicting dyspnea on exertion, chest size with infundibular diameter in systole and diastole were found to be significant. When used appropriately, they were found to correlate in 70 per cent of the cases in determining the presence or absence of dyspnea and in 60 per cent in determining the degree of dyspnea. The simultaneous prediction of presence, absence and degree correlated in only 50 per cent of the cases.

Slightly better results were calculated for the prediction of squatting, using chest size with main pulmonary artery and aortic diameters as significant predictors. Presence or absence of cyanosis correlated in 77 per cent and severity in 62 per cent, using the main pulmonary artery diameter and degree of aortic overriding as significant predictors. The presence of clubbing correlated in 60 per cent and severity in 65 per cent, using infundibular diameter in systole, main pulmonary artery diameter, and aortic to pulmonary artery density ratio as predictors.

Hemoglobin concentration predictions were within 3 gm. in 66 per cent of the cases. One-half of those not correlating were below 14.8 gm. per cent, and one-third were greater than 21.0 gm. per cent.

Kjellberg *et al.*⁸ describe the dextroposi-

tion of the aorta as due to the ventricular septum presenting mainly in a frontal plane with the right ventricle in front of the left. Other authors^{6,11} describe the dextroposition to be more functional than anatomic. We found aortic overriding to be absent or minimal in 70 per cent of all cases. It was insignificant as a predictor for all factors except cyanosis.

The evidence suggests that although there may be true overriding (partial transposition of the aorta) in certain cases, in most instances the overriding of the aorta on the ventricular septal defect is functional in origin. The position of the ventricular septal defect in relation to the aortic root and the enlargement of the aorta secondary to the increased flow in this vessel tend to create the appearance of overriding in those cases where the anatomic position of the aorta is normal. Overriding of the aorta represents a minor factor only in the production of the clinical syndrome of tetralogy of Fallot, and in a minority of cases only. Of much more importance is the high resistance to flow through the obstructed infundibulum and pulmonary valve into the pulmonary artery, and the resultant right to left shunt through the ventricular septal defect.

The occurrence of right aortic arch with tetralogy of Fallot is more than coincidence. However, this *in vivo* study utilizing selective angiocardiology shows a much lower incidence than previously found with conventional roentgenographic examination, barium swallow, or previously reported autopsy series.

SUMMARY

Forty-seven cases of tetralogy of Fallot undergoing selective angiocardiology were reviewed. Computer techniques were employed in calculating equations from the angiocardiology findings for predicting the future clinical course of tetralogy patients.

Right aortic arch was found to be less prevalent (8 per cent) than previously reported.

Overriding of the aorta was of minor degree in 71 per cent of all cases. It was found to be of minor importance in determining the clinical course of patients.

Almost one-half (43 per cent) of all patients were found to have noncardiac birth anomalies or a family history of congenital heart disease.

Pulmonary valvular stenosis was present in 35 per cent of all patients utilizing angiocardiology criteria for diagnosis.

Prediction equations for the presence of decreased growth, cyanosis, squatting, clubbing, dyspnea, and polycythemia were calculated utilizing various angiocardiology measurements, such as infundibular, aortic, and main pulmonary artery diameters. Seventy per cent accuracy was found when these equations were tested against previously reviewed cases.

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VISUALIZATION OF THE PULMONARY ARTERY AND RIGHT VENTRICLE VIA A PATENT DUCTUS ARTERIOSUS*

REPORT OF 4 ILLUSTRATIVE CASES

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WHEN the ductus arteriosus is patent, an opaque catheter under roentgenoscopic observation may be passed through it following percutaneous retrograde femoral arterial puncture.¹¹ The catheter can then be advanced into the pulmonary artery, through the pulmonic valve, and into the right ventricle. Injection of contrast medium will readily result in visualization of the right ventricle, pulmonary artery, and ductus. Selective injection of each structure may, when indicated, be secured. Electronic electrocardiographic equipment permits monitoring of the heart and instant recognition of arrhythmias. Should cardiac arrest or ventricular fibrillation occur, electrical defibrillators, closed chest massage, mouth to mouth breathing, and artificial oxygenation provide means of resuscitating the patient. These measures and improved contrast agents have significantly reduced the dangers of selective angiocardiology.

Herein is reported the value of angiography of the pulmonary artery and right ventricle in 3 patients with congenitally patent ductus arteriosus, and in a fourth patient in whom a ductus was created artificially (Pott's procedure) in order to alleviate severe cyanosis due to tetralogy of Fallot. In 1 patient, reversal of blood flow in the patent ductus, absence of the right pulmonary artery, and aortic origin of the right pulmonary artery were demonstrated. Ligation of the ductus and pulmonary artery originating from the aorta and re-anastomosis to the pulmonary artery were curative and reduced the pulmonary hypertension to nor-

motensive levels. In a second patient, the diagnosis of corrected transposition of the great vessels with bidirectional shunting across a ventricular septal defect was readily established after selective angiocardiology of both ventricles. A third patient with an atypical murmur of patent ductus arteriosus was also found to have pulmonic regurgitation. Ligation of the ductus was curative with cessation of the murmur. Finally, the patient with the artificial ductus was found to have severe pulmonic stenosis which prevented passage of the catheter beyond the pulmonic valve. Withdrawal of the catheter from the ductus with advancement below the aortic valve resulted in the demonstration of a markedly hypertrophied ventricle.

REPORT OF CASES

CASE 1. *Reversed Patent Ductus Arteriosus, Congenital Absence of Right Pulmonary Artery, and Right Pulmonary Artery Arising from Aorta.* This 4 year old Caucasian male was spontaneously delivered after an uncomplicated pregnancy at another hospital. At the age of 7 weeks when he developed dyspnea and cyanosis, and failed to gain weight, he was transferred to this hospital. On December 14, 1959, an intravenous angiocardiology study showed a patent ductus arteriosus with reversal of blood flow. However, the aortic origin of the right pulmonary artery was not demonstrated because the ascending aorta was not opacified. The patient was digitalized and did well. He was re-admitted for more definitive angiographic studies on February 17, 1964. A chest roentgenogram showed enlargement of the heart, pulmonary artery, and pulmonary vasculature of the right

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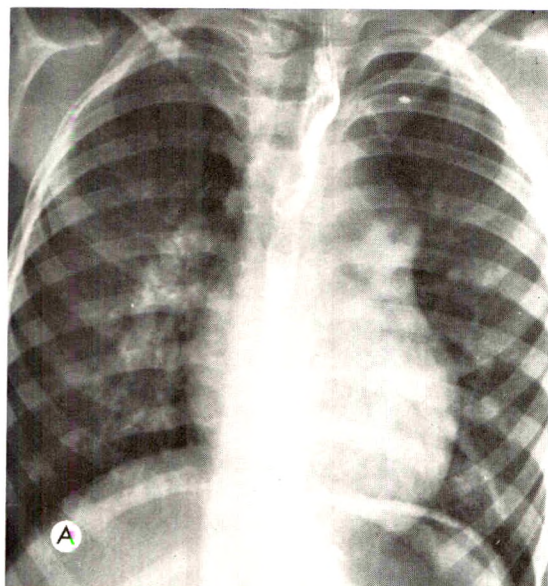


FIG. 1. Case I. *Reversed Patent Ductus Arteriosus with Aortic Origin of Right Pulmonary Artery.* (A) Frontal esophagram showing enlargement and prominence of the heart, pulmonary artery, and right lung vasculature.

lung (Fig. 1A). He was well developed and well nourished. A systolic murmur (Grade 2 to 6) was heard along the left sternal border radiating into the second left intercostal interspace. An electrocardiogram showed right ventricular hypertrophy. The hematocrit was 42 per cent.

Percutaneous transfemoral selective aortography permitted the radiopaque catheter to be advanced through a patent ductus arteriosus. It was passed into the pulmonary artery, through the pulmonic valve, and into the right ventricle. Selective angiocardiograms demonstrated a large right ventricle, enlarged main pulmonary artery, absent right pulmonary artery, and reversal of blood flow through a patent ductus arteriosus (Fig. 1, B and C). The catheter was then withdrawn from the right heart structures and ductus arteriosus, and advanced beyond the aortic arch and valves into the left ventricle. Selective angiocardiographic studies demonstrated an aberrant right pulmonary artery arising from the ascending aorta (Fig. 1, D and E).

On June 22, 1964, Dr. S. Frank Redo ligated the patent ductus arteriosus and anastomosed the anomalous right pulmonary artery to the main pulmonary artery trunk, utilizing a Teflon graft. The patient made an uncomplicated recovery. When last seen in the clinic on

September 2, 1964, he was well and asymptomatic. Only a soft early systolic murmur (Grade 1-2) was heard along the left sternum.³

CASE II. *Patent Ductus Arteriosus and Transposition of the Great Vessels.* At birth at another hospital, a Caucasian male infant was cyanotic and so was transferred to this center. On admission on November 25, 1953, there was cardiac enlargement, plethoric lungs, and a narrowed appearance of the great vessels at the base of the heart. He was discharged unimproved on December 5, 1953, and re-admitted because of heart failure 6 months later. After digitalization he improved. On March 12, 1961, at the age of 7 years, he was admitted for angiocardiographic studies which showed corrected transposition of the great vessels, inversion of the ventricles, and a bidirectional ventricular septal defect.

He was re-admitted, at the age of 10 years, on March 5, 1964, because of dyspnea and cyanosis. On examination, there was intense cyanosis of the skin and mucous membranes. The patient was short (132 cm.) and weighed 23.9 kg. There was bulging of the left precordium and the heart was enlarged. A loud systolic (Grade 5) murmur was heard along the left sternal border radiating to the back. The femoral pulses were regular and strong. The blood pressure was 90/60 mm. Hg. The hemoglobin was 23 gm./100 ml. and the hematocrit was 69 per cent. The electrocardiogram showed incomplete right bundle branch block, right and left ventricular hypertrophy, and probable enlargement of both atria. A conventional frontal roentgenogram showed enlargement of the heart and plethoric lungs (Fig. 2A). On March 6, 1964, a percutaneous transfemoral aortogram demonstrated a large patent ductus arteriosus (Fig. 2B). The catheter was then advanced via the pulmonary artery into a ventricle which was located along the left border of the cardiac silhouette. Early opacification of the aorta also indicated a right-to-left shunt at the ventricular level (Fig. 2, C and D). The catheter was then withdrawn from the posterior ventricle, pulmonary artery, and ductus and passed upward into the arch, ascending aorta, and into a ventricle. Angiocardiograms demonstrated a large trabeculated ventricle which occupied the anterior and right border of the cardiac silhouette (Fig. 2, E and F). The centrally placed pulmonary artery and left hilar location of the ascending aorta were in the classic positions of

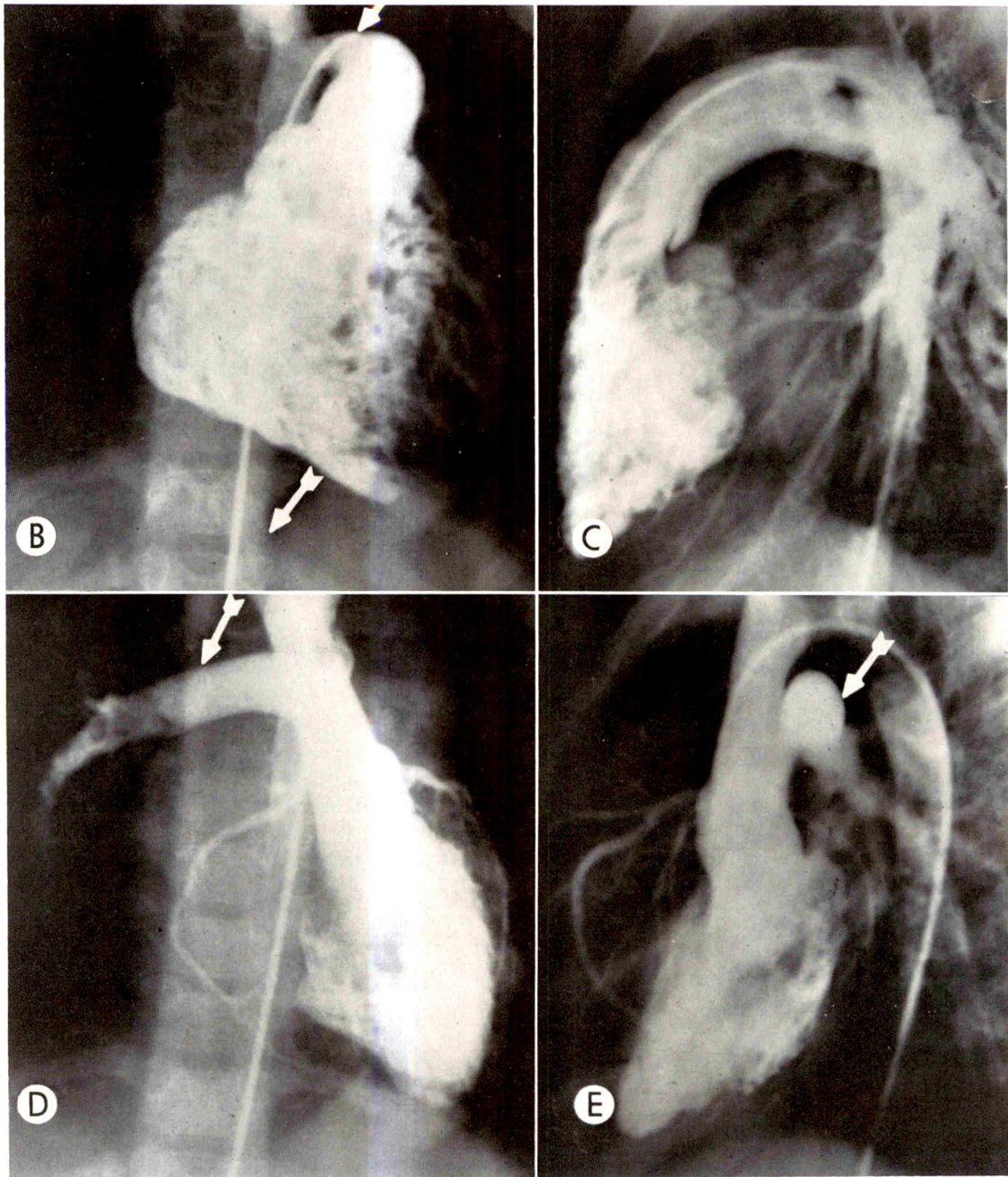


FIG. 1. (B) Frontal angiogram via a catheter percutaneously passed in retrograde fashion from a femoral artery through a patent ductus arteriosus (upper arrow) into the right ventricle. Note the enlarged trabeculated right ventricle, cluster of pulmonary vessels in the lower left lung field, and opacification of the descending aorta (lower arrow). Opacification of the descending aorta from the right ventricle indicates that blood flow is reversed in the ductus. Also note absence of the right pulmonary artery. (C) Biplane lateral view of B. The anterior location of the right ventricle and pulmonary artery identifies these structures and rules out transposition of the great vessels. (D) Selective angiogram obtained by opacifying the heart via a catheter which was advanced over the aortic arch, below the aortic valve and positioned in the left ventricle. Note the large right pulmonary artery (arrow) arising from the aorta. (E) Biplane lateral study of D. The right pulmonary artery (arrow) is "on end" and arises from the posterior border of the ascending aorta.

corrected transposition of the great vessels (Fig. 2, C and E).

CASE III. Patent Ductus Arteriosus with Pulmonic Valvular Regurgitation. A 10 year old Caucasian female was admitted because of a recently discovered murmur during a routine examination. A systolic murmur followed by a prolonged diastolic murmur (Grade 2 to 6) was heard in the second left interspace. The peripheral pulses were normal and the blood pressure was 100/65 mm. Hg in the arms. An electrocardiogram was normal. Roentgenoscopy showed a normal sized heart, increased pulmonary vasculature, and a pulsatile pulmonary artery (Fig. 3A). Because a late systolic crescendo murmur was not heard or seen in the phonocardiogram, aortography was ordered. Percutaneous transfemoral aortography was performed on April 13, 1964, and the catheter was advanced through a patent ductus arteriosus into the pulmonary artery just above the valves. An angiogram revealed insufficiency of the pulmonary artery (Fig. 3B). The right ventricle, thoracic aorta, and the structures at the root of the aorta were normal. The ductus was ligated and the murmur ceased; the patient's recovery was uneventful.

CASE IV. Artificial Patent Ductus, Pulmonic Stenosis, Transposition of the Great Vessels, and Single Ventricle. A 14 year old Caucasian boy was referred for angiography. He had been cyanotic at birth and at the age of 7 months, at

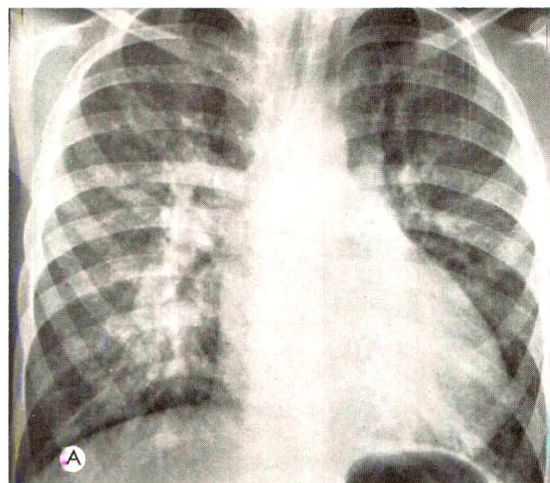


FIG. 2. Case II. Patent Ductus Arteriosus and Transposition of the Great Vessels. (A) Frontal esophagram showing cardiac enlargement and plethoric lungs.



FIG. 2. (B) Retrograde aortogram showing a large patent ductus arteriosus (arrow).

another hospital following the diagnosis of tetralogy of Fallot, an artificial aorticopulmonary shunt (Pott's procedure) was created. Following this, he was well but missed 30 days per year of schooling because of recurrent upper respiratory infections. Physical examination showed cyanosis; however, with good nourishment and development. The blood pressure was 110/50 mm. Hg. A continuous murmur (Grade 4) was heard along the left sternal border and left hemithorax. The hemoglobin was 13.9 gm./100 ml. The electrocardiogram showed sinus tachycardia, first degree heart block, runs of supraventricular tachycardia, right atrial and ventricular enlargement, and right ventricular strain.

Cardiac catheterization, on October 8, 1963, disclosed identical pressures in the aorta and right ventricle, 114/63 and 114/63 mm. Hg, respectively. The pulmonary artery could not be entered with the catheter despite extensive maneuvering. The aorta was, however, readily catheterized. Percutaneous transfemoral aortography on October 10, 1963, permitted entrance of a catheter through a ductus but the catheter could not be advanced into the cardiac

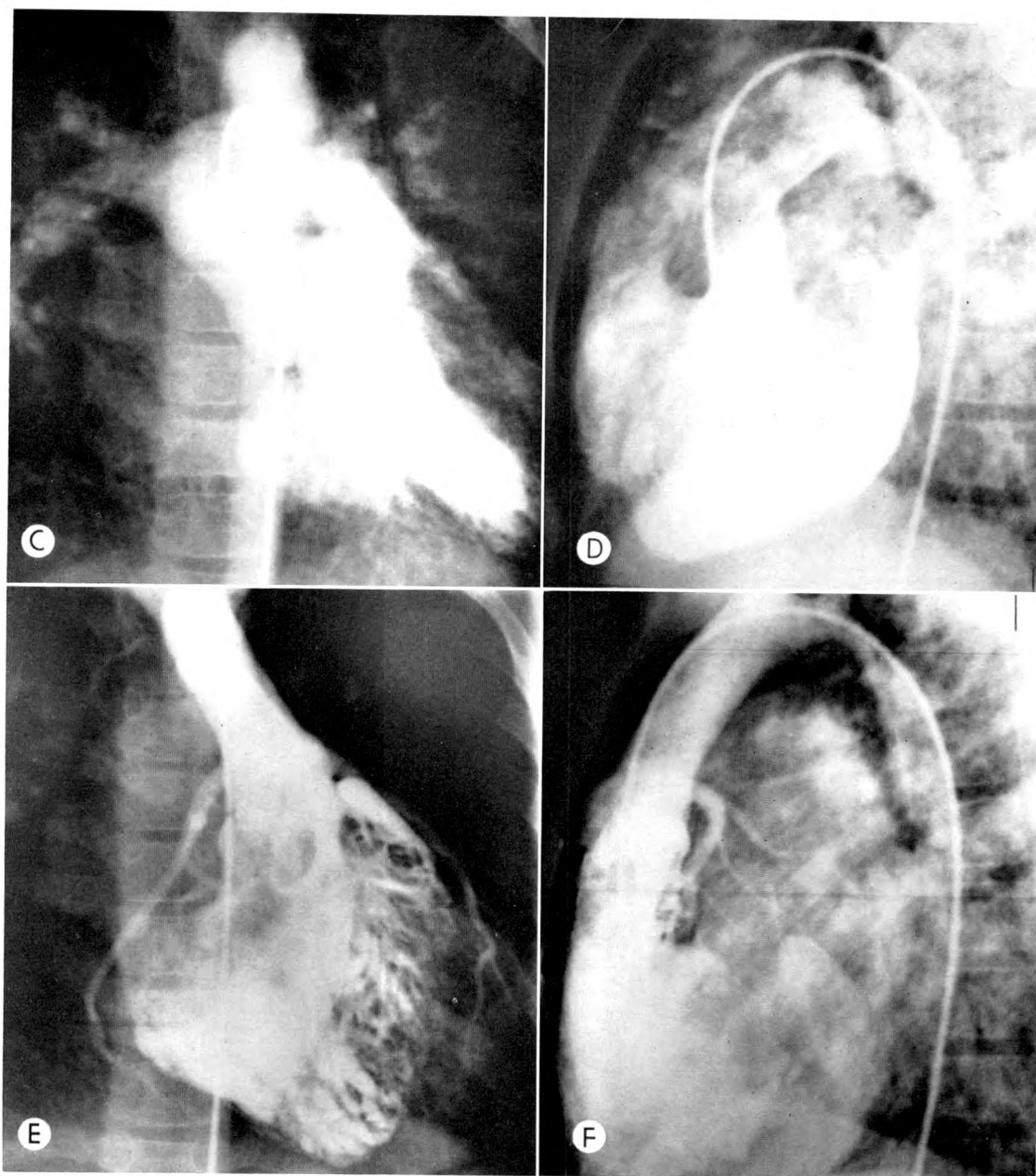


FIG. 2. (C) Frontal angiogram made by advancing catheter from femoral artery, through ductus arteriosus, pulmonary artery, and into the cardiac chamber. Note the smooth walled ventricle, centrally placed and enlarged pulmonary artery and branches, and simultaneous opacification of the aorta and opposite ventricle. (D) Biplane lateral angiogram of C. Note the posterior location of the ventricle beneath the pulmonary artery and the position of the aorta anterior to the pulmonary artery—classic features of transposition of the great vessels. (E) Frontal angiogram made by pulling the cardiac catheter through the ductus arteriosus, advancing it over the aortic arch and below the aortic valve into the ventricle. Note the large trabeculated appearance of the ventricle and opacification of the opposite cardiac chamber. (F) Lateral biplane view of E. The aorta is anterior in the classic location of transposition of the great vessels location.

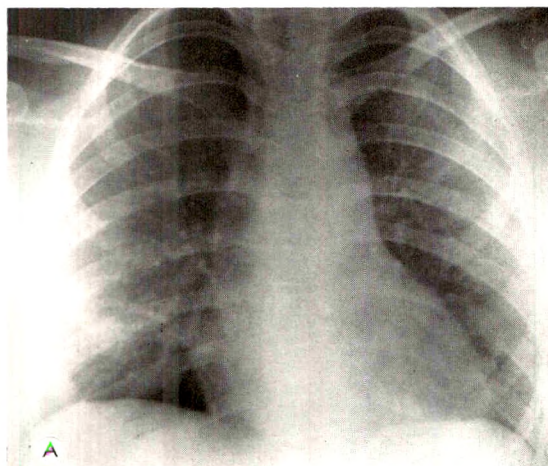


FIG. 3. Case III. *Patent Ductus Arteriosus and Pulmonic Regurgitation.* (A) Frontal teleroentgenogram showing prominence of the pulmonary artery and slight plethora of the pulmonary vasculature.

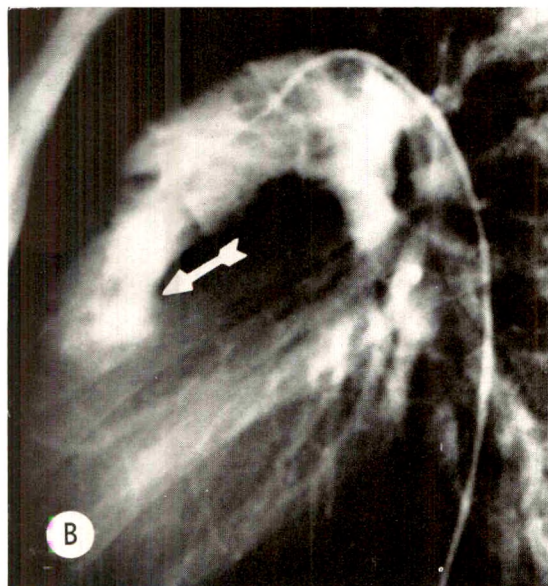


FIG. 3. (B) Lateral angiocardigram made by injecting contrast medium just proximally to the pulmonic valve via a catheter that was advanced from the femoral artery, aorta, and patent ductus arteriosus. Note streaming of contrast medium into the pulmonary conus (arrow).

chamber. Suprapulmonic valve injection of contrast material resulted in the demonstration of severe pulmonic stenosis (Fig. 4, A, B, C and D). The catheter was then withdrawn from the pulmonary artery and advanced over the arch of the aorta, through the aortic valve, and into

a ventricle. A large ventricle with the trabeculated appearance of a right chamber was visualized (Fig. 4, E and F).

DISCUSSION

Mudd and associates⁵ have recently reviewed the literature^{2,4,6,7,13} and added 2 cases of right pulmonary artery originating from the aorta. Systemic arteries arising from the aorta when there is absence of a pulmonary artery, especially bronchial arteries, are common and have been described in the literature.^{8,9,10,12} Case I, however, is of special interest because pulmonary hypertension had caused reversal of blood flow in the ductus arteriosus (Fig. 1, B and C). Caro, Lermenda and Lyons,² in 1957, apparently were the first to ligate a ductus, and re-anastomose an aberrant pulmonary artery arising from the aorta with the pulmonary artery. Their patient, however, succumbed after the operation. The patient reported by Armer *et al.*¹ and Case I appear to be the first to be successfully operated on.

Visualization of both sides of the heart with the technique described above clearly established the diagnosis of transposition of the great vessels and demonstrated a bidirectional ventricular septal shunt in Case II (Fig. 2, C, D, E and F). In a third case, the cause of an unusual murmur heard in a patient suspected of having a patent ductus arteriosus was demonstrated by the injection of a contrast agent proximal to the pulmonic valve by advancing the catheter through the patent ductus. Pulmonic valvular incompetence (Fig. 3B) was also demonstrated. Ligation of the ductus apparently corrected the pulmonic regurgitation, for the murmurs ceased following ligation of the ductus. In a fourth case, severe pulmonic stenosis was roentgenographically demonstrated by injection of contrast medium through a cardiac catheter that had been passed from the femoral artery, through an artificially created aortopulmonary shunt (Pott's procedure), into the pulmonary artery (the valve could not be traversed by the cath-

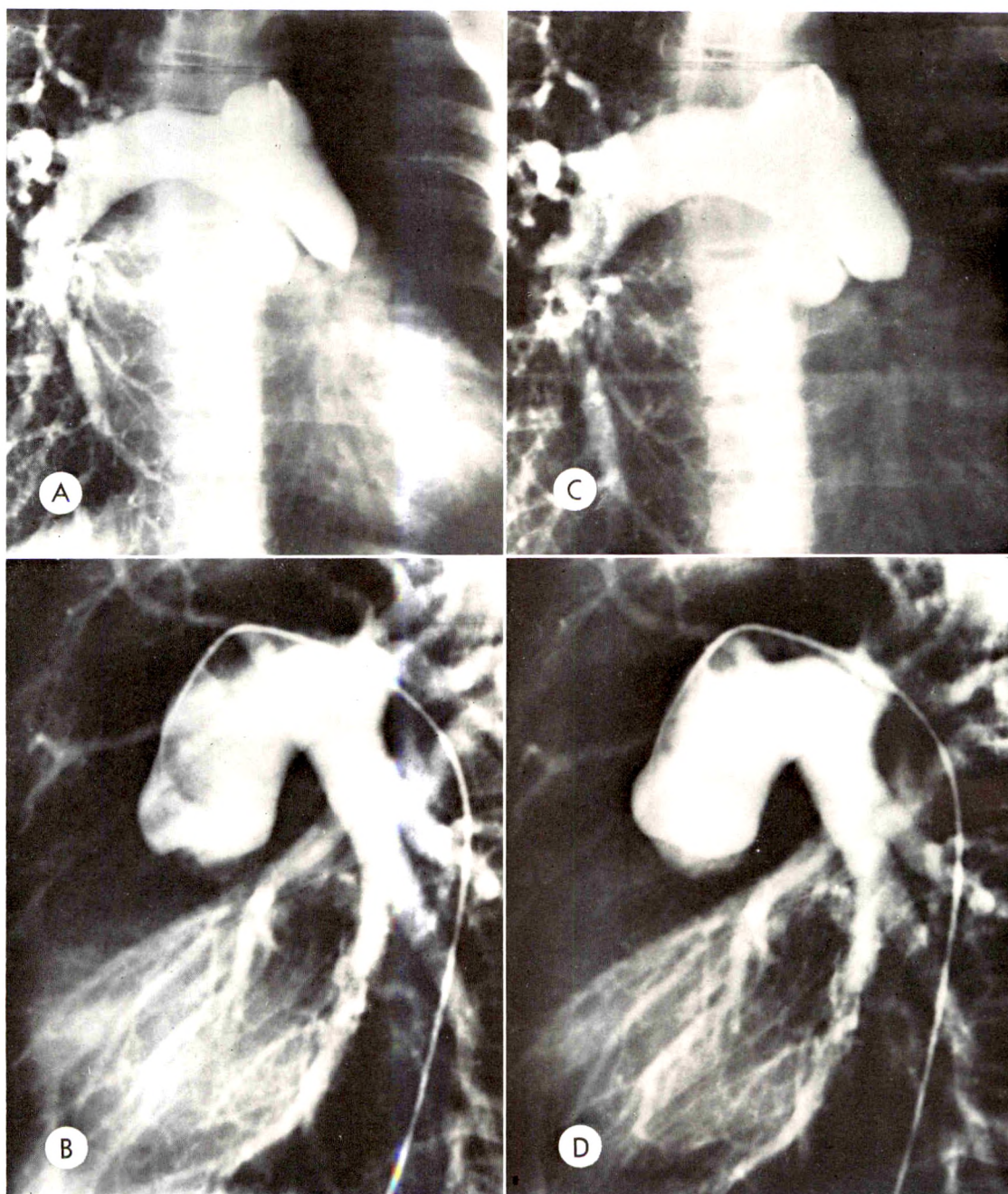
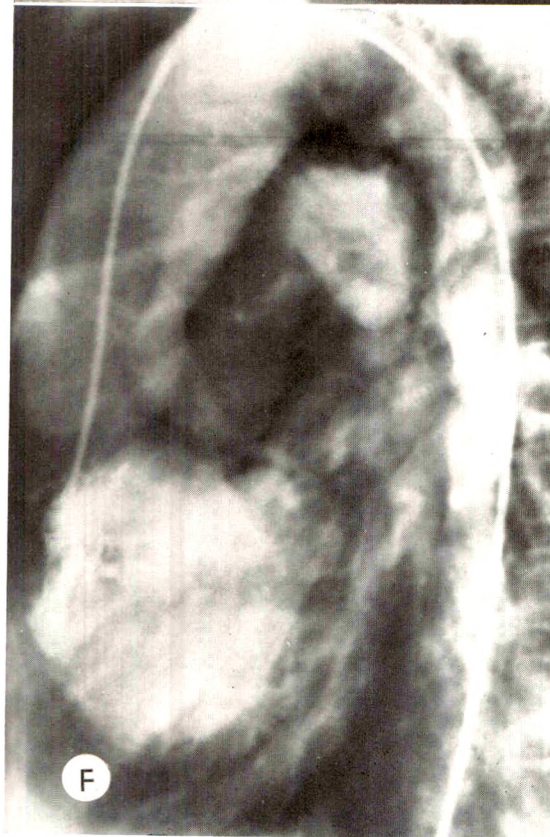
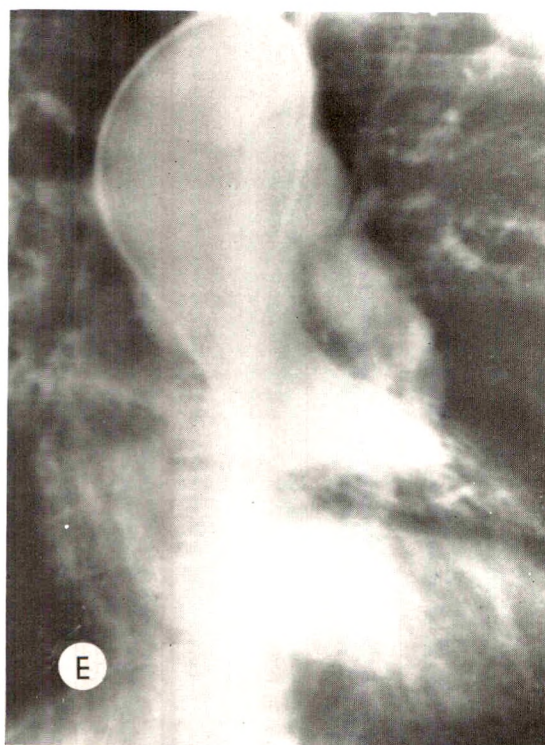


FIG. 4. Case IV. *Pulmonic Stenosis Demonstrated by Supravalvular Injection of Contrast Medium into an Artificial Aorticopulmonary Shunt.* (A) Frontal selective pulmonary artery angiogram during pulmonic systole showing tight immobile pulmonic cusps. Note that aorticopulmonary blood flow is predominantly into the right pulmonary artery. (B) Biplane lateral view of A. (C) During pulmonic diastole (in frontal view) the thickened immovable cusps are evident. (D) Biplane lateral view of C.



ter) (Fig. 4, A, B, C and D). The demonstration of transposed great vessels and a single ventricle precluded surgery.

SUMMARY AND CONCLUSIONS

When a ductus arteriosus is patent, a radiopaque catheter may be passed into the pulmonary artery and right ventricle in order to visualize these structures. Ventricular septal defects, transposed great vessels, absence of a pulmonary artery, pulmonic regurgitation and/or stenosis may be readily diagnosed. If the catheter is then withdrawn into the descending aorta and advanced over the aortic arch and root into the left ventricle, ventricular septal defects with left-to-right shunts and left atrial regurgitation may be recognized. A supra-avalvular injection of contrast medium will also rule out aortic valvular regurgitation or stenosis, aortic root lesions (such as aorticopulmonary windows, aortic sinus aneurysms, bicuspid aortic valves and supra-avalvular stenosis), and transposition of the great vessels. Thereby, other lesions complicating patent ducti arteriosi whether in the right or left side of the heart can be diagnosed, and aid in the treatment of the patient.

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ADDENDUM

Since this paper was accepted for publication another patient with a patent ductus arteriosus had visualization of the cardiac chambers and great vessels via a counter current injection of contrast material into the left brachial artery. A 2½ month old infant, born prematurely, weighing 2.8 kg., in heart failure, and with murmurs of a ventricular septal defect and patent ductus

FIG. 4. (E) Frontal-selective angiogram made by opacifying large ventricle below aorta. (F) Biplane lateral view of E.

arteriosus was studied on March 9, 1965. An 18-gauge Lindeman needle was inserted into the left brachial artery via cut-down. Injection of 5 ml. of 75 per cent hypaque with a Gidlund pressure apparatus (5 kg./cm.²) visualized the patent ductus which was 8 mm. in length and 3 mm. in width. There was mild pulmonic regurgitation. Plethoric pulmonary arterial and venous branches, an enlarged left atrium, ventricle, and aorta were opacified. A left-to-right shunt, at the level of the membranous portion of the left ventricular septum, was demonstrated. Later in the series, the right ventricle and enlarged pulmonary artery was revisualized.

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ROENTGENOGRAPHIC MANIFESTATIONS OF ENDOCARDIAL FIBROELASTOSIS*

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ENDOCARDIAL fibroelastosis, a condition characterized by an abnormal fibroelastic proliferation, manifests itself as a grayish or pearly-white thickening of the endocardium. Its presence hinders the normal contractility and distensibility of the heart. The condition is termed primary if it occurs alone or secondary if associated with other cardiac anomalies. It is the purpose of the authors to describe the roentgen manifestations of this entity.

PATHOLOGY AND HEMODYNAMICS

In primary endocardial fibroelastosis the significant pathologic lesion is located within the endocardium itself; the endocardium of the left ventricle and left atrium is thickened to a variable degree, sometimes measuring up to 2 mm. The left ventricular chamber is usually enlarged (so-called Type I), but rarely may be of normal size or contracted (so-called Type II).² In both types there is diminished contractility and distensibility of the left ventricle and, in addition, incomplete emptying and hypertrophy of this chamber. The result is an obstruction to the flow of blood into the left ventricle which, in turn, is responsible for the right heart enlargement commonly present in patients with endocardial fibroelastosis.

The process may affect the chordae tendineae with resultant thickening and shortening. It may also involve the papillary muscles. Moller *et al.*³ noted that the papillary muscles to the anterior leaflet of the mitral valve arose high on the left ventricular wall and that the chordae to this

leaflet were thickened, shortened and more horizontal than normal—features which result in incompetency of the mitral valve.

In secondary endocardial fibroelastosis, the endocardial thickening is variable in its extent and location and is often less extensive than in the primary form. It is found predominantly on the left side when associated with left-sided obstructive lesions such as coarctation of the aorta and aortic stenosis; it is also found on the left in patients with anomalous origin of the left coronary artery from the pulmonary trunk. It may also occur in the right ventricle in some cases of right ventricular outflow obstruction.

The etiology of endocardial fibroelastosis is unknown. Fetal endocarditis and an enzymatic defect in the cardiac musculature have each been considered as a possible cause of the primary form of the disease. Recent studies at this institution strongly implicate an intrauterine mumps infection.⁴ Abnormal pressures and intracardiac flow currents have been suggested as a possible cause of secondary fibroelastosis.¹ The invariable presence of endocardial thickening in the distribution of the anomalous left coronary artery from the pulmonary trunk suggests that anoxia of the myocardium may be a significant factor.

MATERIAL AND METHODS

This paper is based on a study of 56 patients at the University of Minnesota Hospitals with endocardial fibroelastosis who died during the period 1949–1962. Of these patients 22 were considered primary;

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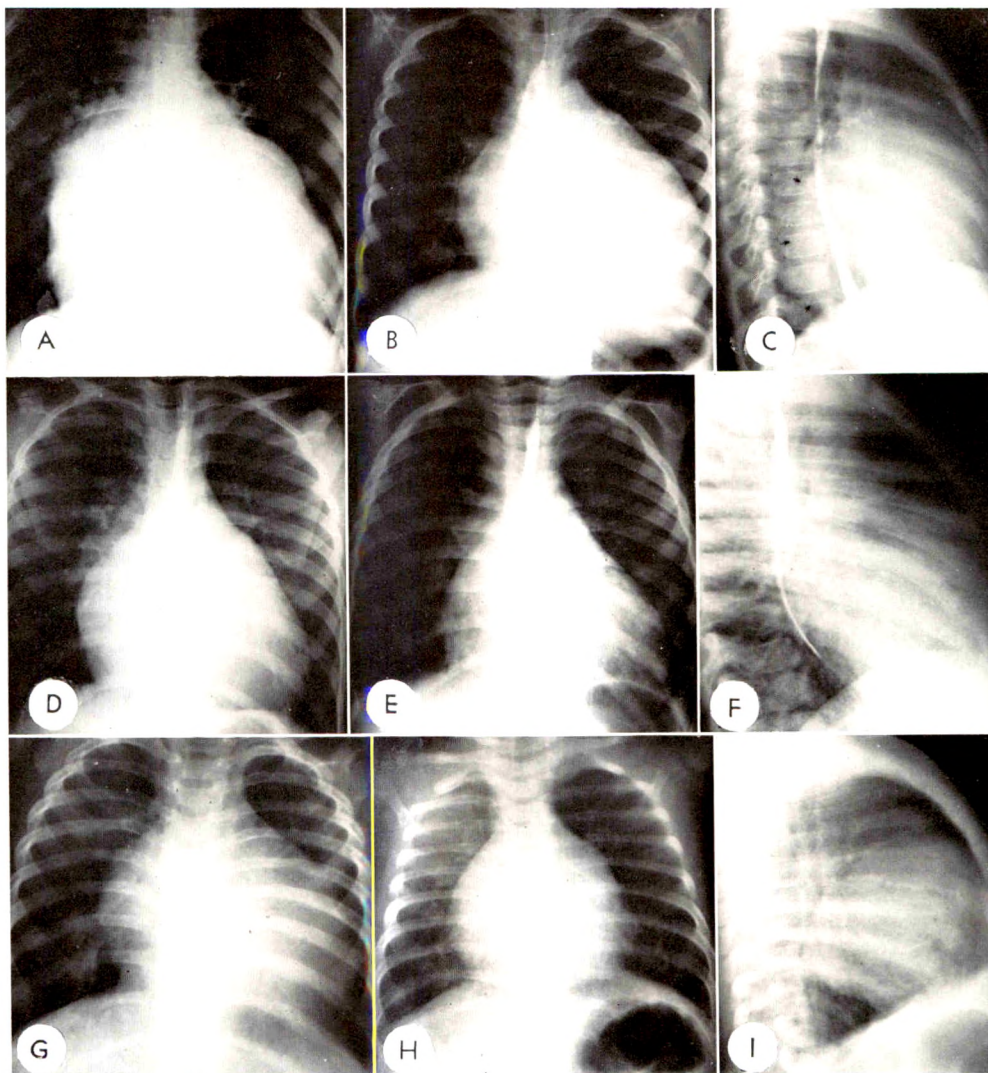


FIG. 1. Series of frontal and lateral roentgenograms of the thorax of patients with primary endocardial fibroelastosis.

A. Male, aged 9½ years. Generalized cardiomegaly with striking enlargement of all chambers.

B and C. Female, aged 7 years. Frontal and lateral views with "esophageal escape" demonstrated in lateral view. Necropsy showed left atrial and left ventricular enlargement.

D. Female, aged 16 years. Frontal view showing left atrial enlargement and moderate enlargement of the right atrium.

E and F. Male, aged 4 years. Frontal and lateral views showing a long segment esophageal deformity caused by combined left atrial and left ventricular enlargement.

G. Male, aged 20 months. Cardiomegaly with the unusual feature of a normal sized left atrium (Figure 7, C-F shows angiocardiograms).

H and I. Female, aged 7 months. Frontal and lateral views of thorax with rare Type II form of primary endocardial fibroelastosis with normal sized left ventricle and enlarged left atrium and right ventricle (Fig. 5, A-E shows angiocardiograms).

21 of these were classified as Type I (15 females and 6 males) and 1 patient, a female, exhibited a contracted left ventricle and was classified as Type II. The age of

onset of symptoms ranged from birth to 5 years and the age at death from 2 days to 16 years.

Thirty-four of the 56 cases of endocardial

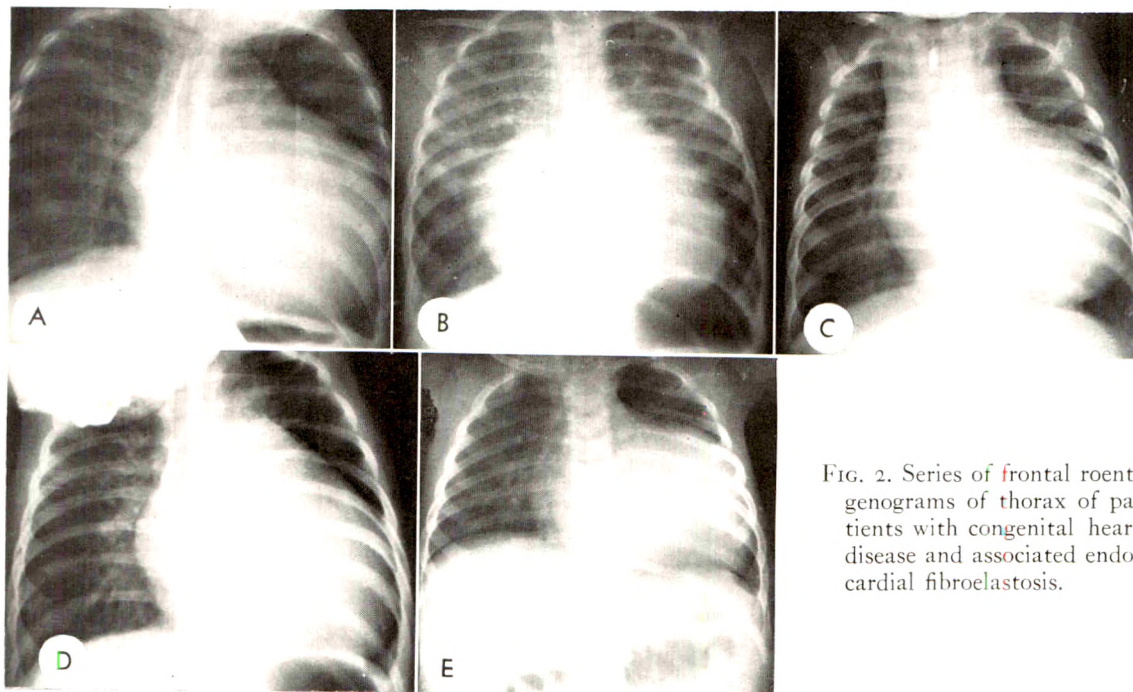


FIG. 2. Series of frontal roentgenograms of thorax of patients with congenital heart disease and associated endocardial fibroelastosis.

A-C. Three different patients with combined aortic stenosis and coarctation.
 D. Anomalous origin of left coronary artery from the pulmonary trunk.
 E. Glycogen storage disease.

fibroelastosis were associated with some other congenital cardiac malformation and were thus examples of secondary fibroelastosis. There were 22 males and 12 females. The onset of symptoms ranged from birth to 14 months and the time of death from 2 days to 3 years. Twenty-four (approximately 70%) patients had abnormalities of the left ventricular outlet or the proximal aorta, such as subaortic stenosis, anomalous coronary artery, valvular aortic stenosis, coarctation of the aorta and hypoplastic proximal aorta (Table 1). In these patients the endocardial fibroelastosis primarily affected the left-sided chambers and their valves. Both left and right-sided ventricular endocardial involvement was seen in 4 patients; in 1 of these there was, in addition, right atrial involvement and in another tricuspid valve involvement. Right-sided involvement alone occurred only once; this was in a patient with a patent ductus arteriosus and left-to-right shunt.

ROENTGENOGRAPHIC FINDINGS IN PRIMARY ENDOCARDIAL FIBROELASTOSIS

Roentgenograms of the thorax of 22 patients were reviewed. The most striking finding on the conventional studies in most patients was the presence of moderate to marked cardiac enlargement and normal or near normal appearance of the pulmonary vasculature. Cardiomegaly was invariably present. Thirteen patients showed marked enlargement, 7 had moderate and 2 exhibited mild cardiomegaly.

Enlargement of the heart was not limited to the left chambers; in 14 patients there was a variable prominence of the right heart border on frontal view suggesting right atrial enlargement. In 16 patients there was evidence of right ventricular enlargement in the lateral view. One patient exhibiting a contracted left ventricle showed striking right atrial and right ventricular enlargement.

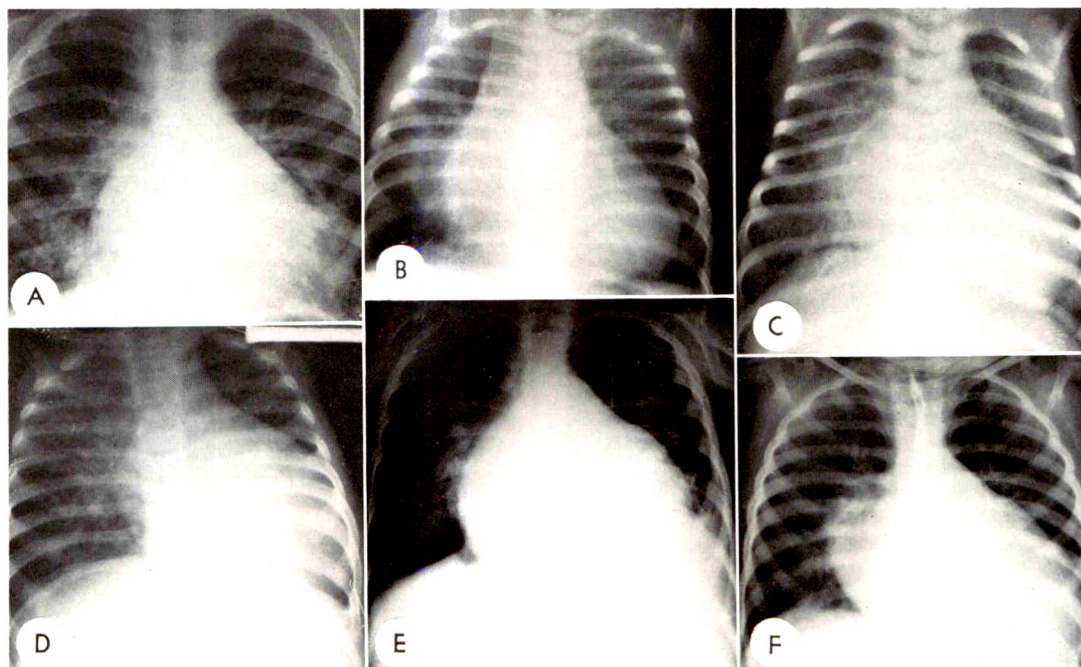


FIG. 3. Series of frontal roentgenograms of thorax of patients with endocardial fibroelastosis demonstrating pulmonary findings.

- A. Pulmonary congestion due to heart failure.
- B. Pneumonitis of right middle lobe.
- C. Pneumonitis and pulmonary congestion.
- D. Atelectasis of left lower lobe (see Figure 4, E and F).
- E and F. "Fusion" of congested hilar vessels with right cardiac border simulating pneumonitis.

TABLE I

NATURE OF ASSOCIATED LESIONS IN 30 PATIENTS
WITH SECONDARY ENDOCARDIAL FIBROELASTOSIS

Isolated Coarctation of Aorta	2
Isolated Aortic Stenosis	6
Combined Aortic Stenosis and Coarctation of Aorta	2
Coarctation of Aorta and Congenital Mitral Stenosis	1
Coarctation of Aorta and Patent Ductus Arteriosus	3
Aortic Stenosis, Subaortic Stenosis and Patent Ductus Arteriosus	1
Isolated Patent Ductus Arteriosus	1
Hypoplastic Left Heart Syndrome*	5
Origin of Anomalous Coronary Artery from Pulmonary Trunk	4
Glycogen Storage Disease	1
Ventricular Septal Defect	2
Eisenmenger's Complex	1
Atrioventricularis Commune	1

* Syndrome characterized by hypoplastic left ventricle, aortic stenosis, hypoplastic aorta and patent ductus arteriosus.

The appearance of the pulmonary parenchyma varied. In the absence of clinically demonstrable cardiac failure, the parenchyma appeared within normal limits. However, pulmonary congestion was demonstrated roentgenographically at some stage of the disease in 14 patients. When the hilar vessels were engorged, the vessels on the right side sometimes obscured the right heart border, "fusing" with it to give a false impression of a right middle lobe pneumonitis (Fig. 3E). This appearance was present in 7 patients. Pleural effusion was noted in 5 cases.

Contrast studies were done on 6 patients. Forward angiocardiology was performed in 5 patients. All showed a normal sequence of opacification with no evidence of an intra- or extracardiac shunt. Slow emptying of the right ventricle and pulmonary arteries, suggesting poor cardiac output and

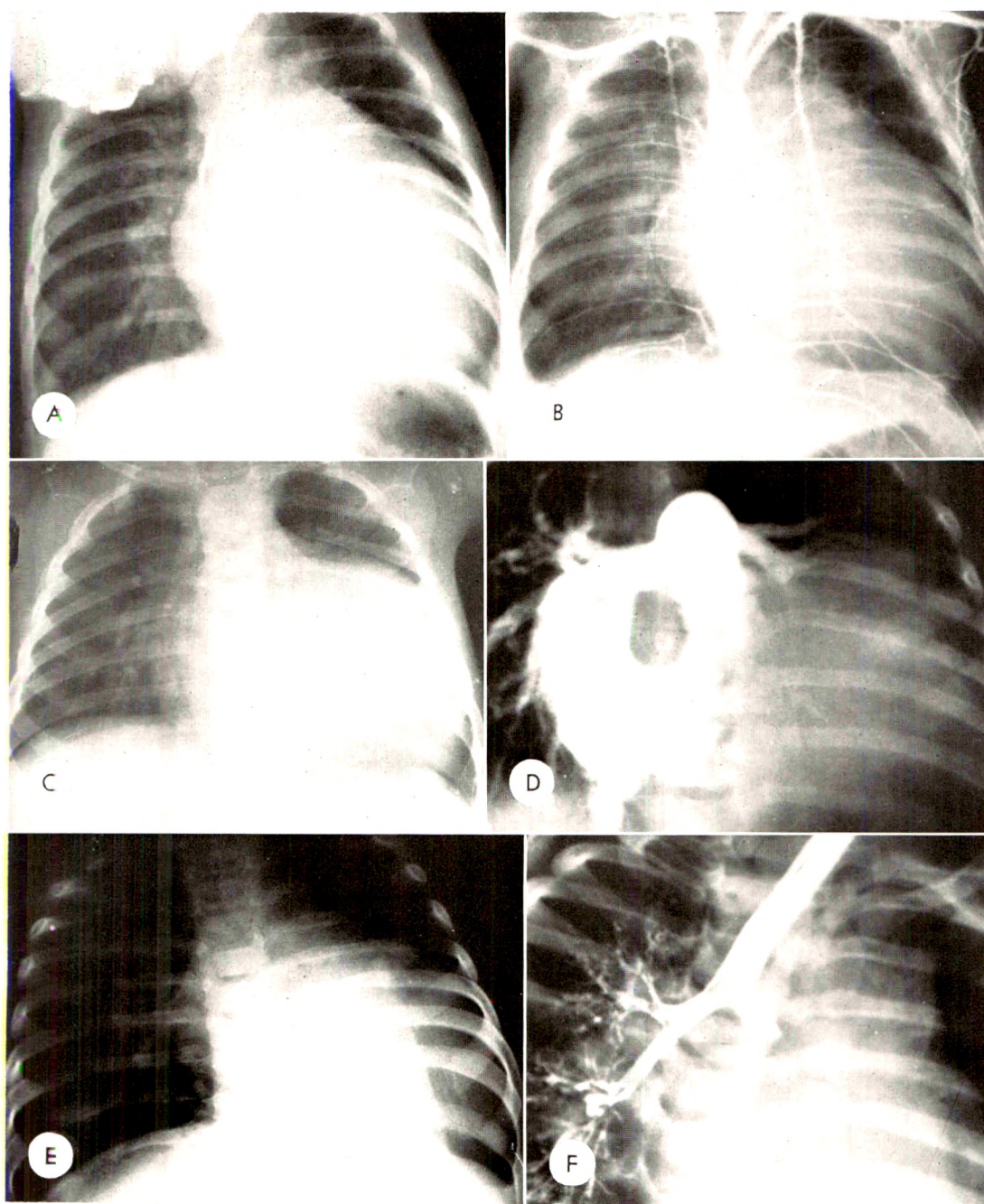


FIG. 4. *A* and *B*. Frontal roentgenogram of thorax and retrograde aortogram, respectively, of a 10 week old female patient with anomalous origin of left coronary artery from the pulmonary trunk and endocardial fibroelastosis. Roentgenograms show displacement of esophagus and aorta by enlarged cardiac chambers.

C and *D*. Frontal roentgenogram and forward angiogram of a 6 month old boy with glycogen storage disease and endocardial fibroelastosis. Roentgenograms show compression of pulmonary arteries to the left lower lobe and compression of the right ventricle by a grossly enlarged left ventricle.

E and *F*. Frontal roentgenogram of the thorax and bronchogram of a patient with endocardial fibroelastosis showing left lower lobe atelectasis caused by compression of the left main stem bronchus by an enlarged left atrium.

obstruction to flow from the left side of the heart, was noted in 1 patient (Fig. 5, *A* to *E*). Left ventriculography in 2 patients showed a striking and persistent opacification of the left atrium, indicating the presence of mitral insufficiency (Fig. 6, *A* to *F*). Both types of procedures provided information concerning the size and contractility of the left atrium and left ventricle. Five of the 6 patients showed a dilated, hypertrophied, poorly contracting left ventricle; the findings were striking in 3 and mild to moderate in 2 patients. One patient exhibited a near normal sized left ventricle with fairly normal appearing contractions; this patient represented the only example of the so-called Type II variety of primary fibroelastosis (Fig. 5*D*). An enlarged, poorly contracting left atrium was observed in 5 patients; the remaining case atypically demonstrated a normal sized atrial chamber (Fig. 7, *D* and *E*).

ROENTGENOGRAPHIC FINDINGS IN SECONDARY ENDOCARDIAL FIBROELASTOSIS

Roentgenograms of the thorax were available for review in 30 of the 34 patients with secondary endocardial fibroelastosis. The associated congenital defects in this group are shown in Table 1. Moderate to marked cardiomegaly was present in 28 patients. At the time of the initial roentgenographic studies, the diagnosis of endocardial fibroelastosis was included in the differential diagnosis in 16 patients because of the size and configuration of the heart, the suggestion of left sided enlargement and normal appearing pulmonary vasculature.

Contrast studies were carried out in 13 patients. Satisfactory biplane contrast examinations were available for review in 6 patients. Left ventriculography was performed in 2 patients. In 1 of these, the study demonstrated diminished contractility of the left ventricle and mitral insufficiency; in addition, a peculiar configuration of the left ventricle associated with subaortic stenosis was demonstrated (Fig. 7,

A and *B*). In the other patient, left ventriculography demonstrated prolonged opacification of a smooth-walled, enlarged left ventricle but evidence of mitral insufficiency was absent (Fig. 8, *E* and *F*).

Aortography was performed in 5 patients and demonstrated coarctation in 1, patent ductus arteriosus in 1, aortic stenosis in 2 and an anomalous coronary artery arising from the pulmonary trunk in 1 patient.

Forward angiocardiology was performed in 3 patients; the correct diagnosis was suggested in 2 of these because of prolonged opacification of an enlarged, smooth walled left ventricle. In the other patient a left to right shunt was demonstrated, but the diagnosis of fibroelastosis was not suspected.

Of the 30 cases of secondary endocardial fibroelastosis with roentgenograms 20 (66 per cent) had some form of obstruction to the left ventricle, either due to coarctation, aortic stenosis, a combination of coarctation and aortic stenosis or a hypoplastic left ventricular malformation (Table 1).

COMMENT

Roentgenograms of the thorax in patients with endocardial fibroelastosis usually show moderate to marked cardiomegaly. The cardiac configuration in lateral and left anterior oblique views often suggests that the enlargement is left sided. Enlargement of the right heart, commonly present, is frequently unsuspected roentgenographically. Left atrial enlargement is nearly always present and usually causes compression and displacement of the esophagus; marked enlargement may cause compression of the left main stem bronchus, leading to atelectasis of the left lower lobe. Rarely, the enlarged left atrium, in frontal view, may displace the esophagus far to the right while in lateral view the esophageal deformity is minimal; the esophagus appears to "escape" from the cardiac bulk (Fig. 1, *B* and *C*).

The pulmonary vasculature usually appears normal, but during periods of cardiac

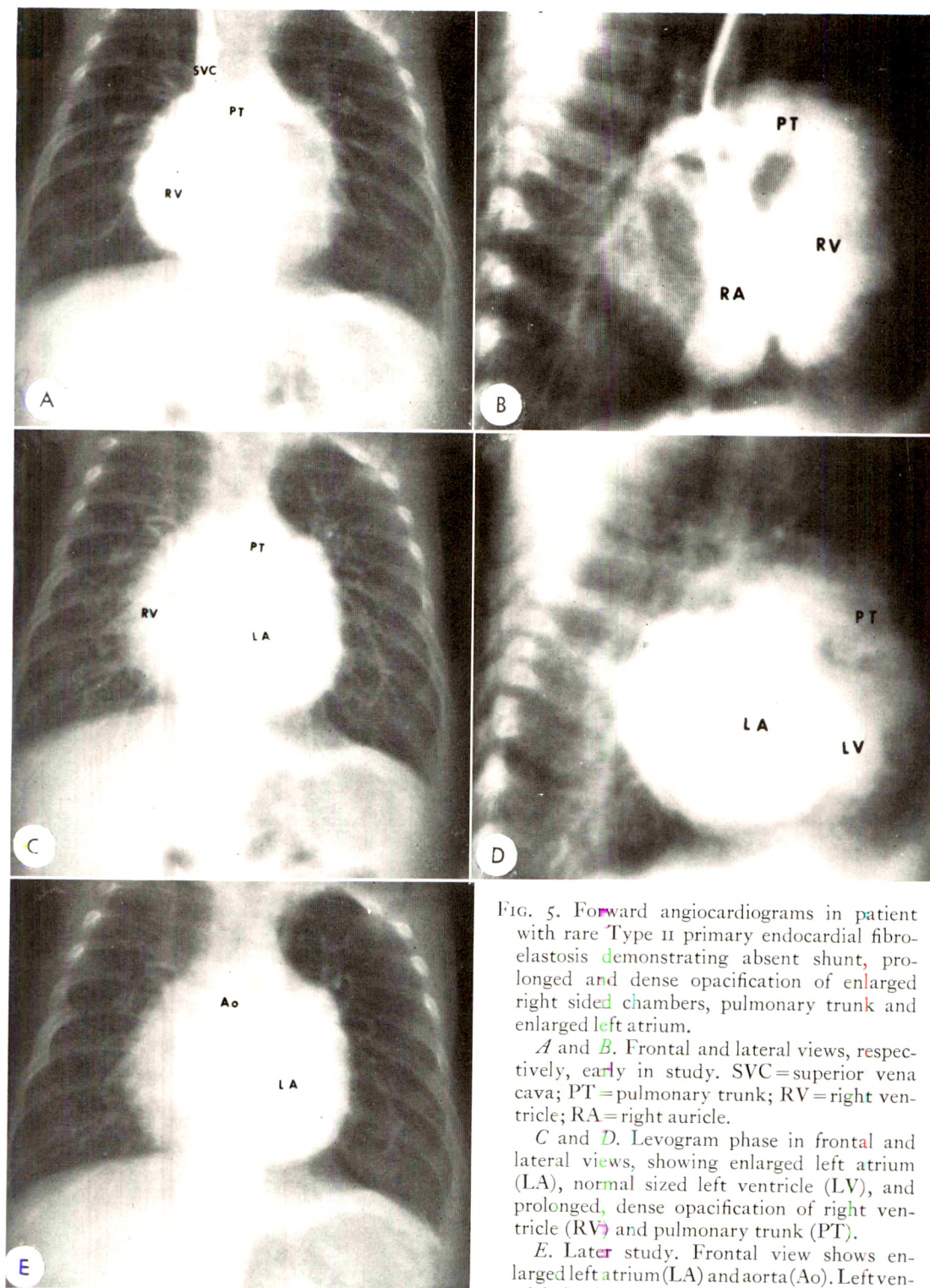
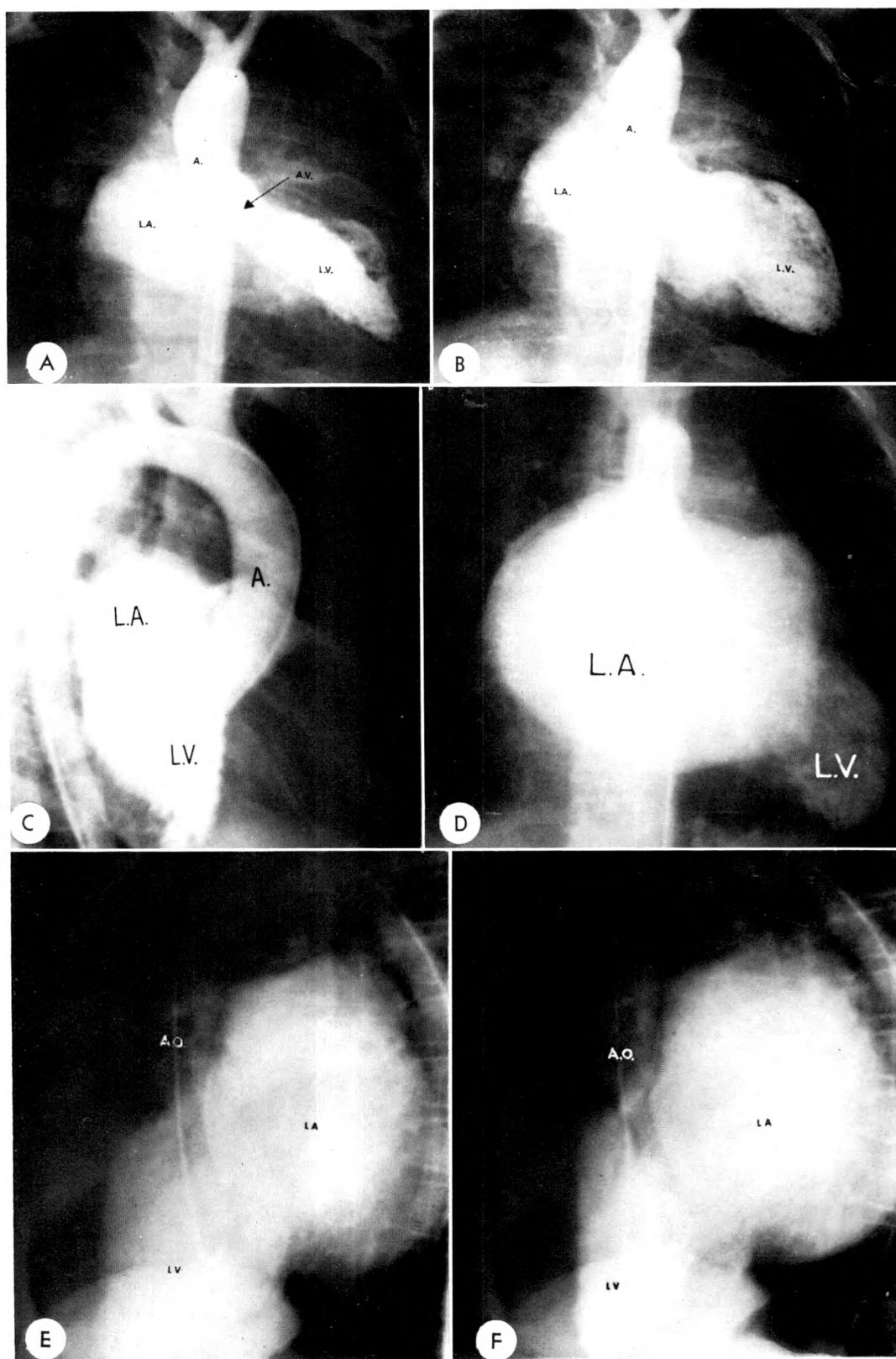


FIG. 5. Forward angiograms in patient with rare Type II primary endocardial fibroelastosis demonstrating absent shunt, prolonged and dense opacification of enlarged right sided chambers, pulmonary trunk and enlarged left atrium.

A and B. Frontal and lateral views, respectively, early in study. SVC=superior vena cava; PT=pulmonary trunk; RV=right ventricle; RA=right auricle.

C and D. Levogram phase in frontal and lateral views, showing enlarged left atrium (LA), normal sized left ventricle (LV), and prolonged, dense opacification of right ventricle (RV) and pulmonary trunk (PT).

E. Later study. Frontal view shows enlarged left atrium (LA) and aorta (Ao). Left ventricle is obscured by the enlarged left atrium.



decompensation pulmonary congestion is observed.

Forward angiocardiography demonstrates a delayed but otherwise normal sequence of opacification of the cardiac chambers in the primary form of the disease and in those secondary forms without shunts. In addition, enlargement of the left atrium and left ventricle may also be shown; the latter chamber may exhibit decreased contractility and a thickened myocardium with a smooth inner wall. In several cases measurements of the diameter of the left ventricular chamber were made during systole and diastole, and it appeared that the largest chambers showed the smallest change in size.

Selective ventriculography will readily demonstrate the presence and degree of mitral insufficiency which is usually present; only 1 patient (with secondary endocardial fibroelastosis) in whom contrast

studies were done showed no evidence of mitral insufficiency (Fig. 8, *E* and *F*).

The findings on conventional studies are not specific for primary and secondary endocardial fibroelastosis. There are other conditions which appear similar roentgenographically and which require differentiation. For example, patients with anomalous origin of a coronary artery from the pulmonary trunk often show electrocardiographic and vectorcardiographic signs of myocardial ischemia; glycogen storage disease may be identified by muscle biopsy. Cardiomegaly due to myocarditis may gradually diminish as the condition resolves; this is not a feature of endocardial fibroelastosis. Mitral valvular disease of rheumatic origin may be impossible to differentiate from fibroelastosis in the absence of a history of rheumatic fever; the age of the patient should be taken into consideration since rheumatic valvular disease is extremely



FIG. 6. Mitral insufficiency in primary endocardial fibroelastosis.

A-C. Left ventriculograms of a 4 year old patient. Frontal and lateral views showing enlarged, smooth walled, left ventricle (LV), moderately enlarged left atrium (LA) and mitral insufficiency. Contractility of both chambers is demonstrated.

D-F. Left ventriculograms in a 16 year old patient. Frontal and lateral views showing marked enlargement of left atrium (LA) and left ventricle (LV) and mitral insufficiency. *E* and *F*, obtained during ventricular diastole and systole, respectively, show contractility of left ventricle but poor opacification of aorta (Ao), suggesting incomplete emptying of left ventricle.

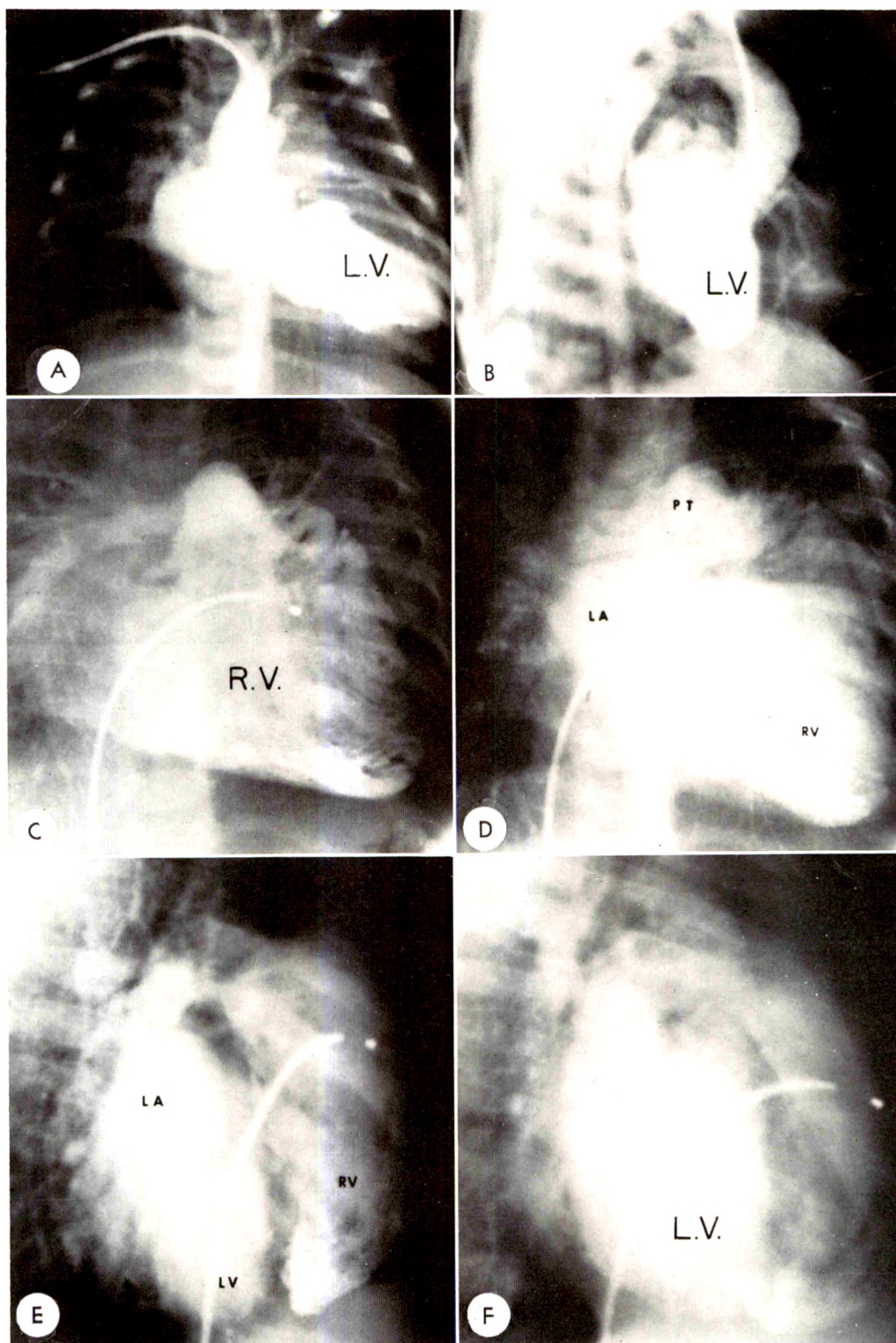


FIG. 7. *A* and *B*. Left ventriculograms of an 8 week old female patient with secondary endocardial fibro-elastosis, subaortic stenosis, valvular aortic stenosis and patent ductus arteriosus. Frontal and lateral

rare in infancy and early childhood. Some other conditions which may be associated with mitral insufficiency include Marfan's syndrome, Hurler's disease and isolated congenital mitral insufficiency.

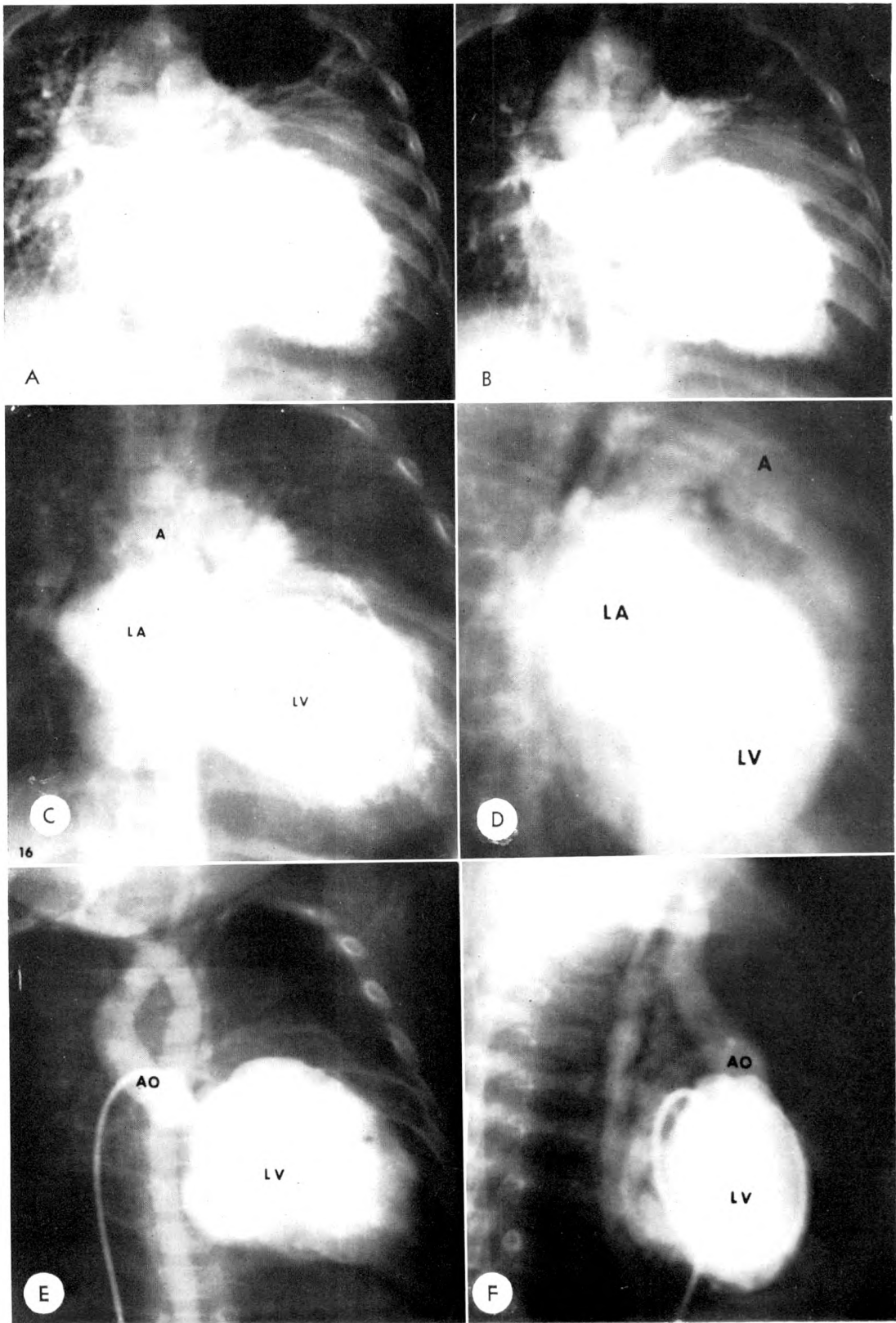
A positive skin reaction to mumps antigen has been demonstrated in patients with both primary and secondary forms of the disease.⁴ A positive test enhances the clinical suspicion of fibroelastosis but cannot serve to differentiate between the two forms of the disease. This being the case and since there is a high incidence of left sided obstructive lesions associated with fibroelastosis, it is our opinion that all cases of suspected fibroelastosis should have angiocardiographic studies, particularly aortography and left ventriculography. Ideally, aortography should be performed selectively by advancing a catheter from

the right brachial or axillary artery; the catheter tip should be positioned just above the aortic valve. In this fashion, the size of the aortic valve ring, the anatomy of the aortic cusps, and the appearance of the supra-valvular portion of the aorta will provide valuable information concerning the functional state of the aortic valve. In addition, coarctation of the aorta or an anomalous origin of a coronary artery from the pulmonary trunk, if present, may be defined. In the latter condition, contrast material flows from the aorta into an enlarged right coronary artery, then through anastomotic branches into a small left coronary artery and, finally, into the pulmonary trunk. In the absence of significant aortic stenosis, the tip of the catheter can often be advanced into the left ventricle; in this way, the size and contractility of this



view, respectively, showing mitral insufficiency. Peculiar configuration of base of left ventricle (LV) in frontal view possibly secondary to subaortic stenosis.

C-F. Right ventriculograms of a 20 month old male patient with primary fibroelastosis. *C.* Frontal view early in study showing striking enlargement of right ventricle. Serpentine appearance of the left lateral margin of the outflow tract of right ventricle (RV) is caused by hypertrophy of muscle bundles. *D* and *E.* Subsequent roentgenograms in frontal and lateral views, respectively. Normal sized left atrium (LA) and the left ventricle (LV) are opacified. The thickened interventricular septum is also shown. *F.* Lateral film exposed 5 seconds after the injection into the right ventricle was completed. Persistent opacification of right ventricle and pulmonary trunk suggesting diminished cardiac output. The enlarged left ventricle is also shown.



chamber and the degree of mitral insufficiency may be defined.

SUMMARY

A study of 56 cases of endocardial fibroelastosis has been made with emphasis on the roentgenographic and angiocardiographic features of the disease.

If unassociated with other forms of congenital heart disease, the condition is classified as primary and preferentially involves the left side of the heart. The left ventricle may be dilated or contracted; the latter form is rare and is associated with a normal sized or contracted left ventricle.

Secondary endocardial fibroelastosis is most commonly associated with coarctation and/or aortic stenosis, and is invariably present when an anomalous left coronary artery arises from the pulmonary trunk.

The important roentgenographic findings are moderate to marked left-sided cardiomegaly and normal appearing lung fields. Right ventricular enlargement is commonly present but often unrecognized in conventional studies.

Angiocardiographic studies will usually reveal an enlarged, smooth walled, poorly contracting left ventricle and insufficiency of the mitral valve.

The use of selective aortography and left ventriculography in all cases of suspected fibroelastosis is recommended in order to confirm the presence of mitral insufficiency, to define obstructive lesions in the aorta and to exclude the presence of an anomalous origin of a coronary artery from the pulmonary trunk.

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The authors wish to express their appreciation to Dr. J. A. Moller and Dr. J. E. Edwards for their interest and guidance during this study.

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FIG. 8. Angiocardiographic findings in secondary endocardial fibroelastosis.

A and B. Forward angiocardiograms of a 6 month old patient with associated glycogen storage disease. Frontal views (A) exposed during atrial systole and (B) atrial diastole showing contractility of the left atrium but little or no contractility of the left ventricle.

C and D. Forward angiocardiograms of a patient with aortic stenosis, coarctation of aorta and endocardial fibroelastosis. Frontal and lateral views, respectively, showing enlarged, densely opacified left atrium (LA) and left ventricle (LV). Smooth outline of left ventricle is well shown in lateral view. Poorly opacified aorta (Ao) suggesting low cardiac output.

E and F. Selective left ventriculograms of a patient with aortic stenosis and associated endocardial fibroelastosis. Frontal and lateral views, respectively, showing a smooth walled, densely opacified left ventricle (LV). Atypically, no mitral insufficiency was demonstrated. Ao = Aorta.

CONGENITAL ABSENCE OF THE LEFT PERICARDIUM*

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ALTHOUGH congenital absence of the pericardium was first described¹ over 400 years ago, there are fewer than 120 cases reported in the literature of the past 250 years. The condition has thus been recognized only on rare occasions in the past and, with few exceptions, most diagnoses of congenital absence of the pericardium have been made incidentally at autopsy or during various thoracic surgical procedures. Since Ellis and his associates⁴ recorded what is probably the first case recognized roentgenographically in 1959, there has been an increasing number of cases reported in the literature.^{1,2,3,5,6,7,8,9} The major purpose of this paper is to emphasize that, although this lesion is rarely recognized, certain features, especially the roentgenographic findings, are sufficiently distinctive to warrant more frequent appreciation.

The deficiency is left sided in the overwhelming percentage of cases reported. Fortunately, the relatively benign condition of complete absence of the left pericardium is more frequent than a partial defect through which the heart may herniate and produce serious symptoms. Right sided lesions and complete absence of the entire pericardium are extremely rare. Associated congenital anomalies are not infrequent, occurring in about 30 per cent of the reported cases. Congenital cardiac defects described include atrial septal defect, patent ductus arteriosus, and bicuspid aortic valve. Other anomalies include bronchogenic cysts, pulmonary sequestration and diaphragmatic hernia. The lesion occurs

predominantly in males in a ratio of about 3:1.

Little indication of the presence of uncomplicated congenital pericardial deficiency can be gained from the patient's history or from physical examination. If herniation and strangulation of the atrium, atrial appendage or ventricle through a partial defect do not occur, pericardial absence apparently does not produce significant symptomatology and a normal life span is to be expected.¹¹ Several young adults whose case histories are available have complained of vague chest pain, presumed in retrospect to be due to torsion of the great vessels secondary to the abnormal mobility of the heart. However, with herniation of the left atrium or left ventricle through a partial pericardial defect, severe chest pain has been described.

Cardiac murmurs, when present, are not specific for the condition. The murmurs are usually systolic and most often heard in the 2nd left intercostal space. They are not the typical ejection murmurs of valvular pulmonary stenosis, a condition which might be suspected from the chest roentgenogram. One finding commonly observed in these patients is bradycardia. Among the many functions attributed to the intact pericardium is its regulation of diastolic cardiac volume, although Moore and Shumacker¹⁰ did not find any cardiac dilatation in 65 dogs subjected to partial or total pericardiectomy. If in the human the pericardium does play a role in containment of cardiac volume, it would seem that in this condition a bradycardia might exist in associa-

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tion with a large stroke volume.

Striking similarities are to be noted in published electrocardiograms in this condition. There is usually marked deviation of the frontal QRS axis in the neighborhood of $+120^\circ$. Fowler⁶ draws the analogy between these electrocardiograms and those taken in normals in the left lateral decubitus body position, thus attributing the electrical changes to abnormal heart position.

ROENTGEN FEATURES

The roentgen features of complete left-sided pericardial defect are sufficiently characteristic to allow identification on routine chest examinations. They may be classified into four categories:

Routine Chest Roentgenograms. Several diagnostic features of complete absence of the left pericardium may be appreciated on a single posteroanterior view of the chest (Fig. 1A; and 6A). There is marked levoposition of the entire cardiac silhouette. This may result in a false impression of cardiomegaly if one fails to recognize the right heart border over the midline or left border of the spine. The contour of the left heart border is abnormally prominent due to accentuation of the convexities of the aortic knob, the main pulmonary artery and the left ventricle. In addition, the space between the aorta and the main pulmonary artery is widened, resulting in sharp demarcation of the pulmonary artery. The pulmonary vasculature is unremarkable. The trachea and esophagus assume a midline position despite the marked levoposition of the aorta. The inferior cardiac border is flattened and slightly elongated and there commonly is interposition of lung between the left dome of the diaphragm and the inferior cardiac border. The lateral view of the chest may show displacement of the cardiac silhouette posteriorly and absence of a clearly defined cardiac border anteriorly (Fig. 1B; and 6B).

Cardiac Fluoroscopy. Fluoroscopy confirms the findings noted on routine studies. There is hypermobility of the heart and, in addition, the cardiac pulsations appear

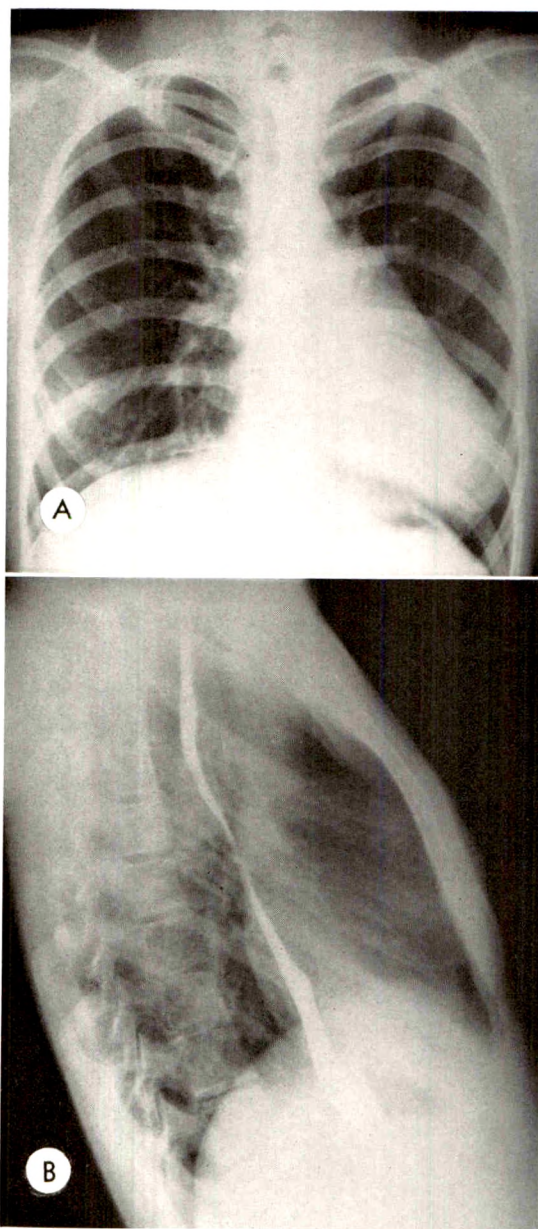


FIG. 1. Case 1. (A) Posteroanterior roentgenogram. Findings include levocardia, prominent left heart border, elongation and flattening of the inferior cardiac border and interposition of lung between the diaphragm and inferior heart border. The trachea and esophagus are in the midline. (B) Lateral roentgenogram. There is displacement of the heart posteriorly and absence of a clearly defined cardiac border anteriorly.

to be of increased amplitude. The prominent pulsations are probably due to the bradycardia often observed in these pa-

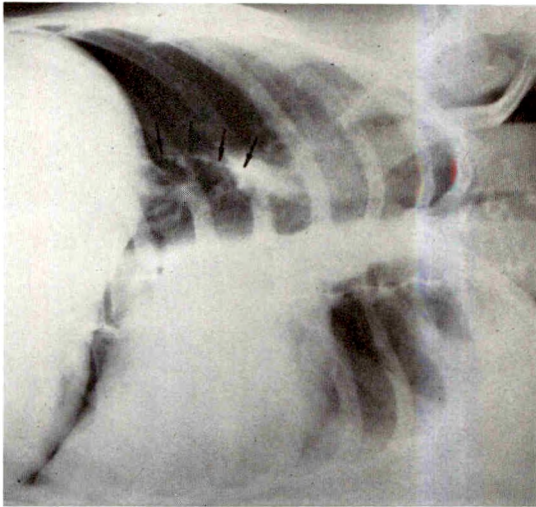


FIG. 2. Case 1. Left lateral decubitus roentgenogram after introduction of 500 cc. of air into the left pleural space. The appearance of a pneumopericardium confirms presence of a pericardial defect.

tients and may also result from the accentuated appearance of the structures comprising the left heart border.

Angiocardiography. This procedure serves the primary function of ruling out other cardiac abnormalities. In addition, it confirms the abnormal cardiac position in a positive manner.

Diagnostic Pneumothorax. The diagnosis of congenital defect of the pericardium may be confirmed by demonstrating a pneumopericardium after the introduction of about 500 cc. of air into the left pleural space (Fig. 2; and 7). This may be seen soon after the induction of the pneumothorax, but occasionally will not be observed until several hours later. Air may also outline the root of the ascending aorta and pulmonary artery. Demonstration of the pneumopericardium requires careful and proper positioning. The ideal position is with the patient lying on the left side in a slight right posterior oblique utilizing a horizontal beam.⁴ This avoids superimposition of the right heart border on the spine and yet allows air to pass freely into the pericardial sac if a defect is present. Roentgenograms taken in inspiration and expiration are valuable while in this position. Failure to out-

line the pericardium with air does not necessarily rule out a pericardial defect as it is possible that adhesions may prevent passage of air over the pericardium.

The pneumothorax may also be utilized to demonstrate other features. An anteroposterior recumbent view will reveal gas between the left dome of the diaphragm and the flattened inferior surface of the heart (Fig. 3; and 8). In addition, upright and supine lateral views of the chest will reveal marked mobility as the heart may be displaced dramatically away from the anterior chest wall (Fig. 4; and 9).

That these findings are sufficiently characteristic to warrant ready recognition of this entity is substantiated in the following case reports.

REPORT OF CASES

CASE 1. A 13 year old white female was admitted to the Mary Fletcher Hospital for evaluation of an "enlarged heart," which was first discovered 4 years prior to admission when chest roentgenograms were taken because of exposure to tuberculosis. She had been asymptomatic until 1 year previously when she noted the onset of exertional fatigue and lightheaded-

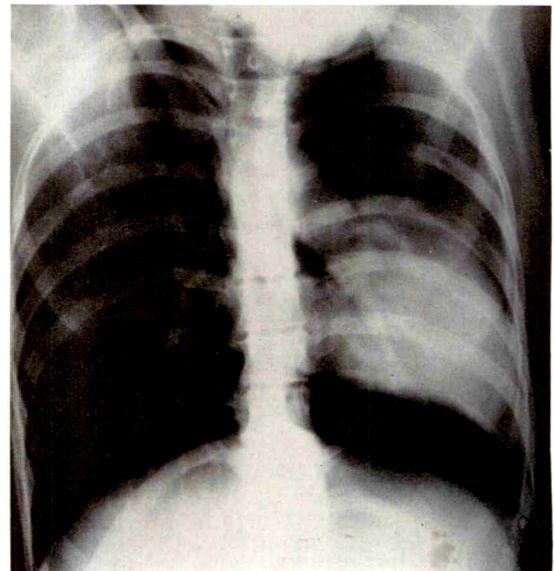


FIG. 3. Case 1. Anteroposterior recumbent roentgenogram after diagnostic pneumothorax. Air passes freely between the base of the aorta and the pulmonary artery as well as beneath the heart.

ness. There was no history of dyspnea, chest pain, cyanosis, or signs of congestive cardiac failure. No past history of rheumatic fever or scarlet fever was obtained. The patient's mother had been pre-eclamptic during this pregnancy, and the patient was born 3 weeks prematurely with a birth weight of 4 lb. 5 oz. Growth and development had been unremarkable and the usual childhood diseases were contracted without sequelae.

On physical examination the heart rate was 50/min., blood pressure 100/58 mm. Hg, respiration 24/min. and temperature 37°C. Positive physical findings were limited to examination of the chest. The point of maximal cardiac impulse was in the 5th left intercostal space 2–3 cm. lateral to the midclavicular line. The second heart sound in the pulmonic area was normally split with normal respiratory variation in the split. A_2 was greater than P_2 . A Grade II/VI short, high-pitched, blowing systolic murmur was heard over the entire precordium, but was loudest in the 2nd left intercostal space near the sternum. There was no associated thrill palpable. The murmur was almost undetectable with the patient in a sitting position. Peripheral pulses were normal, and no clubbing or cyanosis was evident.

The electrocardiogram revealed marked right axis deviation, with terminal conduction defect seen best in leads II, III, and V_5 – V_6 . Right ventricular hypertrophy could not be diagnosed from this tracing. The vectorcardiogram was normal and thus suggested that the electrocardiographic changes were on the basis of the



FIG. 4. Case 1. Supine lateral roentgenogram after diagnostic pneumothorax. The heart is quite mobile and falls posteriorly, considerably away from the sternum.



FIG. 5. Case 1. Exploratory thoracotomy. There is complete absence of the left pericardium.

terminal conduction defect, and perhaps partially due to marked positional changes.

Posteroanterior and lateral roentgenograms (Fig. 1, *A* and *B*) revealed the cardiac shadow to be displaced into the left hemithorax. There was prominence of this left heart border, especially of the pulmonary artery segment. No localized chamber enlargement was evident. Pulmonary vasculature appeared to be within normal limits. The trachea and esophagus were in the midline. There was interposition of lung between the flattened inferior surface of the heart and the left diaphragm. The lateral view revealed displacement of the heart posteriorly and absence of a clearly defined anterior heart border. On fluoroscopy, pulsations of the prominent pulmonary artery segment were not increased, and the aorta was of normal size.

It was decided that simple right heart catheterization should be performed to rule out left-to-right shunts and pulmonary hypertension. This study revealed no evidence of shunt at any level and pressures throughout the right heart and in the pulmonary arteries to be normal. Cineangiocardigraphy showed no abnormality other than levoposition of the heart.

Five hundred cubic centimeters of air were then introduced into the left pleural space by Dr. D. B. Miller. Roentgenograms obtained in the left lateral decubitus position about 1 hour later delineated the right wall of the pericardium (Fig. 2). Air passed freely between the base of the pulmonary artery and the aorta, and also beneath the heart border (Fig. 3). With the patient in the supine position, an

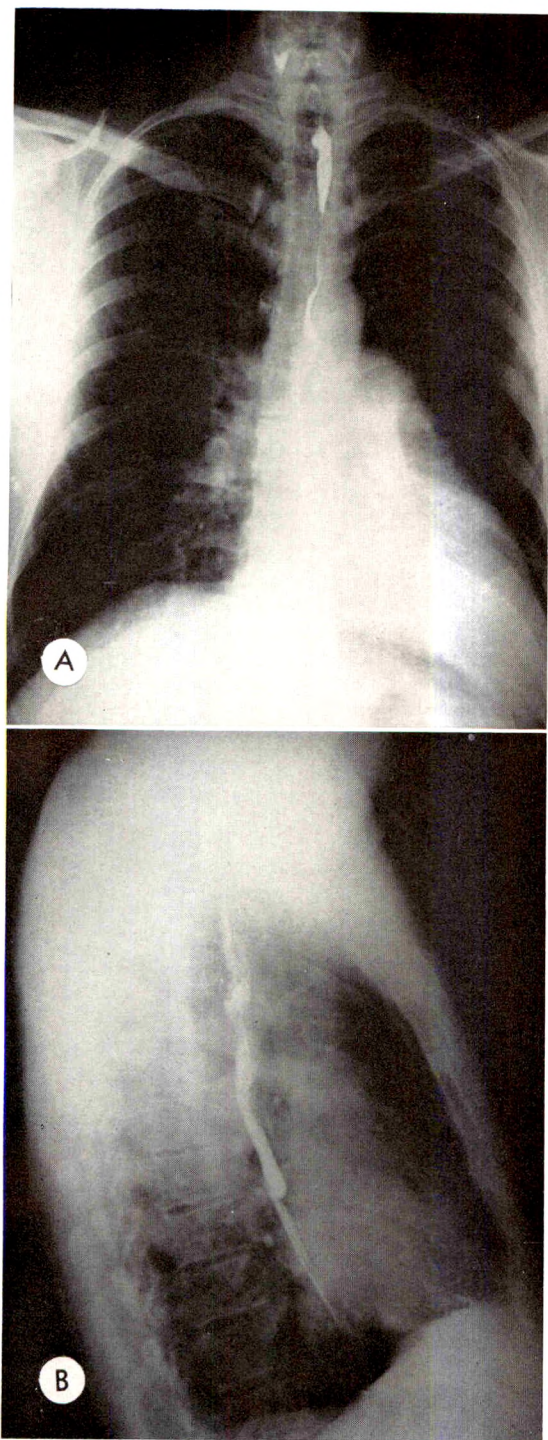


FIG. 6. Case II. (A) Posteroanterior roentgenogram. Findings include levocardia, accentuation of the left heart border (especially the pulmonary artery), prominent pulmonary vasculature, and interposition of lung between the diaphragm and the elongated and flattened inferior heart border.

abnormally large retrosternal space was seen as the heart dropped freely posteriorly (Fig. 4).

An exploratory thoractomy was performed with the intent of creating a left pericardium from parietal pleura. The heart was seen to be freely occupying the left anterolateral aspect of the chest. The heart's contour was altered in that the myocardium had developed a curvature corresponding to the dome of diaphragm. Total absence of the left pericardium without evidence of fenestration through which cardiac herniation might occur was noted (Fig. 5). The major vessels were rotated but otherwise appeared normal. Attempts at surgery to restore the heart to the midline produced immediate and serious drops in blood pressure, possibly by virtue of great vessel torsion. The procedure was therefore terminated, and the patient recovered uneventfully from the surgical intervention.

CASE II. A 53 year old white male farmer was admitted to the Indiana University Medical Center in January, 1962, with the chief complaint of progressive dyspnea on exertion and decreasing effort tolerance. He had been well until age 35 when he first experienced a bout of palpitation. Since then he had evanescent attacks of palpitation and in 1952 he developed exertional dyspnea and mild dizziness. The dyspnea progressively worsened and for the year prior to admission he noted decreasing effort tolerance. Past history included rib fractures at the age of 20 years and an appendectomy in 1955.

Physical examination revealed the heart rate to be 84/min., blood pressure 100/58, respiration 16 and temperature 98.4°F. Positive findings were limited to the chest. The precordium was hyperactive and the maximum cardiac impulse was palpable in the 7th left interspace in the mid-axillary line. There was a Grade 3 systolic murmur over the entire precordium with maximum intensity in the second left interspace. The second heart sound was widely split and fixed in response to respiration. P_2 was increased. An audible third sound was heard at the apex.

The electrocardiogram was very abnormal,

The trachea and esophagus are in the midline. (B) Lateral roentgenogram. The heart is slightly displaced posteriorly and the anterior heart border is poorly defined.

showing evidence of right ventricular hypertrophy and right axis deviation. There was a first degree atrioventricular heart block and marked clockwise rotation. There was also questionable evidence of an old anterior myocardial infarction.

Posteroanterior and lateral roentgenograms (Fig. 6, A and B) revealed marked levoposition of the heart and accentuation of the structures of the left heart border, especially of the main pulmonary artery. The space between the aorta and pulmonary artery appeared unusually wide. The pulmonary vasculature was increased. Interposition of the left lung was prominent between the elongated and flattened inferior heart border and the dome of the left diaphragm. The lateral view revealed the heart to be slightly displaced posteriorly and the anterior heart border to be poorly defined. Fluoroscopy revealed the pulmonary artery pulsations to be increased. Cardiac catheterization and cineangiography revealed the presence of partial anomalous venous return from the right lung into the superior vena cava at the juncture with the right atrium. A sizable left to right shunt was present, but no evidence of pulmonary hypertension was found.

A diagnostic pneumothorax was performed by Dr. E. C. Klatte. This was followed by the appearance of air in the pericardial sac in 1 hour, best demonstrated in the left lateral decubitus view (Fig. 7). In the recumbent position, air could be seen to outline the base of the aorta as well as the inferior heart border (Fig.

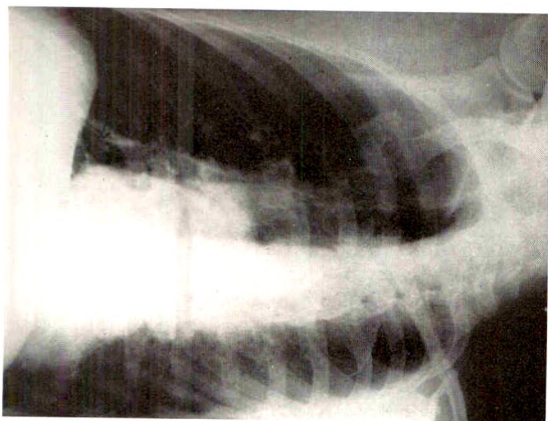


FIG. 7. Case II. Left lateral decubitus roentgenogram after the injection of 500 cc. of air into the left pleural space. The appearance of a pneumopericardium confirms the presence of a pericardial defect.

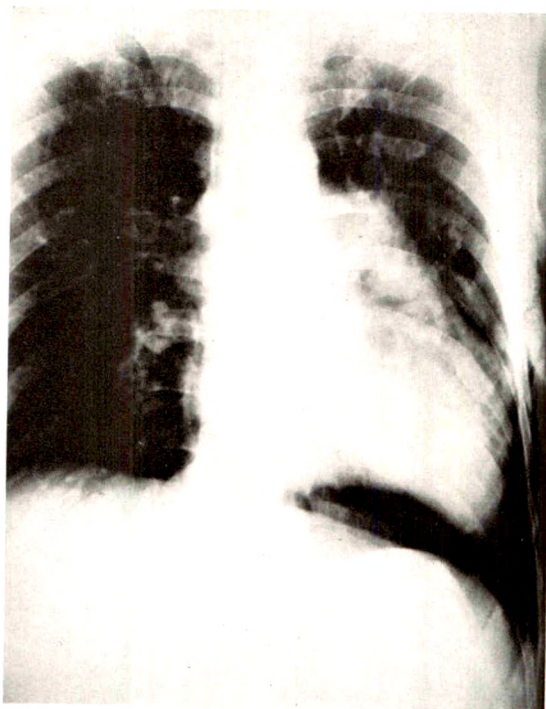


FIG. 8. Case II. Anteroposterior recumbent roentgenogram after diagnostic pneumothorax. Air passes freely between the base of the aorta and the pulmonary artery and beneath the heart.

8). In the lateral position the heart was displaced posteriorly away from the sternum (Fig. 9).

The patient returned the following month for surgery for correction of the anomalous pulmonary venous drainage. Complete absence of left pericardium was confirmed at surgery. The postoperative course was uncomplicated and he was discharged after 7 days. It was noted that the pulse rate had dropped from a preoperative level of 82 to a pulse varying between 32 and 60.

SUMMARY

The clinical and roentgenologic features of congenital complete absence of the left pericardium have been presented along with illustrative case material. The roentgenologic features are sufficiently characteristic to warrant easy recognition and the diagnosis can be confirmed with a diagnostic pneumothorax. However, since the roentgenographic findings are so strikingly similar in the cases reported to date, diagnostic pneumothorax, with its possible

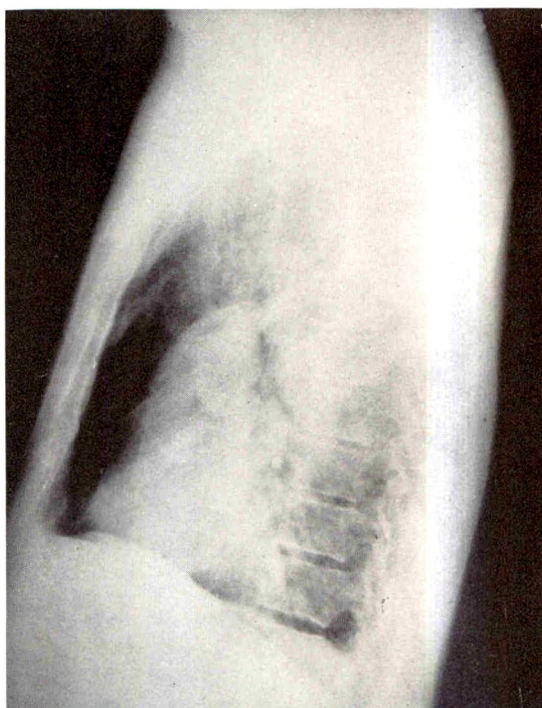


FIG. 9. Case II. Lateral roentgenogram after diagnostic pneumothorax. There is marked mobility of the heart as it falls posteriorly away from the sternum.

hazards, may soon no longer be considered necessary in establishing the diagnosis except in those cases where surgery is considered for correction of a partial defect.

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THORACIC AORTOGRAPHY*

INTRAVENOUS AND SELECTIVE TECHNIQUES

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VISUALIZATION of the thoracic aorta has been successfully performed for over 25 years with the intravenous method of angiocardiology.^{3-5, 8, 13, 16-19, 21-37, 39-42} Because of the coexisting opacification of the left atrium and ventricle after intravenous angiocardiology, visualization of the coronary arterial system, aortic valves, and aortic sinuses has required selective techniques. Supravalvular aortic stenosis, an aorticopulmonary window, and patent ductus arteriosus also require selective aortography for definitive diagnosis.^{6, 7} Even though intravenous angiocardiology allows visualization of the brachiocephalic origins of the great arteries,^{13, 34} the distal portions of the carotid, vertebral, and the entire cerebral circulation are better opacified with direct selective arteriographic techniques.^{9-11, 14, 15, 38, 43} In this paper the value of intravenous thoracic aortography is reiterated and additional data illustrating the value and superiority of selective catheter techniques for diagnosis of disease of the aorta and its brachiocephalic branches are presented.

METHODS AND MATERIALS

Intravenous angiocardiology was performed according to the technique previously published.^{3, 18, 19, 35, 39} In every instance, a preliminary circulation time determination with dilute decholin (sodium dehydrocholate, 20 per cent) was made in order to estimate the time and number of roentgenograms necessary for visualization of the entire cardiovascular system. The Schönander Elema apparatus, usually in a single plane, was used for serial contrast roentgenography. A second examination in an-

other projection was regularly made at the completion of the first series. Selective thoracic aortography was performed via the percutaneous transfemoral or transaxillary approach using the Gidlund apparatus.¹¹ Serial contrast roentgenography was also performed with the Schönander Elema device. The material of this report consists of the presentation of a series of patients with aneurysmal disease of the ascending aorta and/or aortic sinuses, truncus arteriosus, brachiocephalic arterial buckling and/or dilatation, and coarctation of the aorta. Intravenous angiocardiology and selective aortography were used, respectively, for study of cardiovascular disease in these patients.

ILLUSTRATIVE CASES

CASE 1. Dissecting aneurysm of ascending aorta due to medial degeneration²⁹ in a 38 year old man with congenital bicuspid valves of aorta. Sudden onset of excruciating chest pain during a New Year's party required the hospitalization of this patient (at another hospital) for a month. A pericardial effusion spontaneously subsided and the patient became asymptomatic and returned to work. A cardiac murmur had been heard at birth, during school, and military examinations but had not restricted activities of the patient. The heart, especially the left ventricle and ascending aorta, was enlarged several months after onset of chest pain (Fig. 1, A and B). There were systolic and diastolic murmurs at the base of the heart consistent with aortic stenosis and regurgitation. The blood pressure was 118/74 mm. Hg.

Intravenous angiocardiology in the erect seated position and with a single arm injection of concentrated contrast medium on July 1, 1963, disclosed a prolonged circulation time (22 seconds), deformity of the right atrium by

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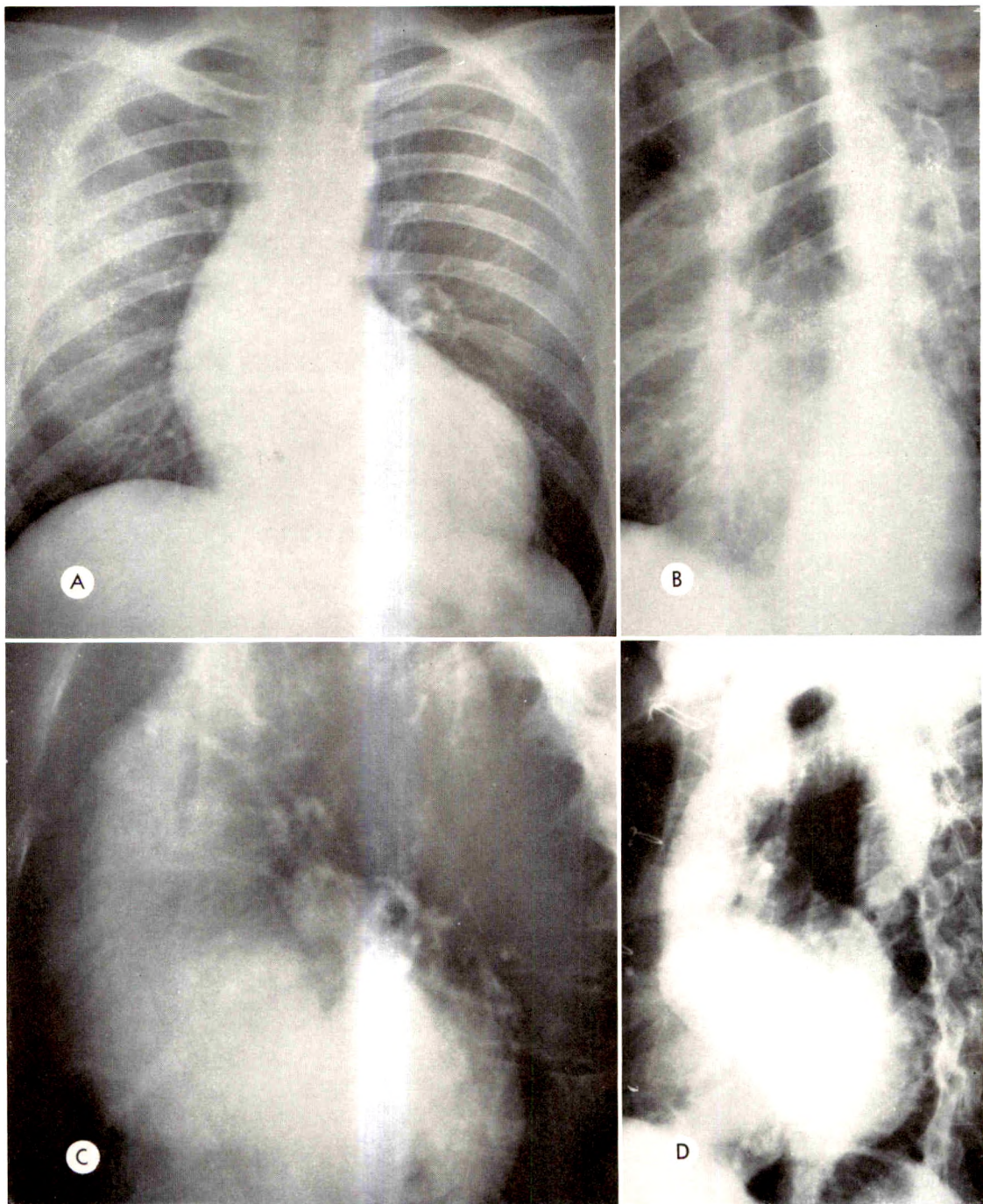


FIG. 1. Case 1. *Intravenous angiocardiology in aneurysmal dilatation of the ascending aorta.* (A) Teleroentgenogram showing enlargement of the left ventricle and ascending aorta. (B) Left anterior oblique roentgenogram of the chest showing the large left ventricle and dilated ascending aorta. (C) Left anterior oblique angiogram revealing the enlarged left ventricle and aneurysmally dilated ascending aorta. (D) Postoperative angiogram showing the dacron graft replacing the aneurysm. (The latter study was made available by Dr. M. E. De Bakey.)

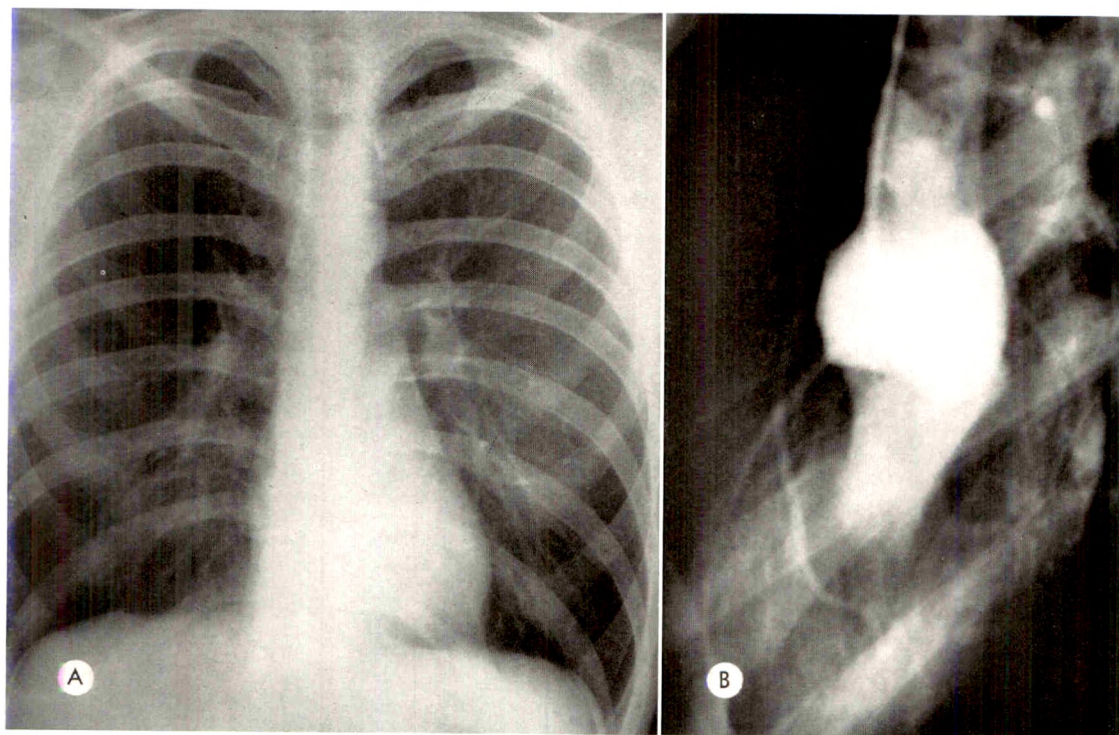


FIG. 2. Case II. Aortic insufficiency and aneurysm of aortic sinuses in Marfan's syndrome. (A) Conventional teleroentgenogram showing a normal heart. (B) Selective aortogram demonstrating aortic regurgitation in a patient with aneurysmal dilatation of the aortic sinuses.

a hugely dilated ascending aorta, and enlargement of the pulmonary artery and branches. The left atrium was also enlarged, while the left ventricular cavity was dilated and the wall enlarged (15 mm.) (Fig. 1C). The ascending aorta was aneurysmally dilated measuring 90 mm. in mid-ascending portion (upper limits of normal, 38 mm.) (Fig. 1C). At operation at another hospital on July 18, 1963, using extracorporeal circulation and coronary perfusion, a dissecting aneurysm of the ascending aorta associated with aneurysmal dilatation of the ascending aorta was found. Bicuspid aortic valves producing moderate aortic insufficiency were also observed. The dissecting aneurysm was excised and replaced with a 30 mm. dacron tubular prosthesis. The patient made an uneventful recovery and intravenous angiocardigraphy, prior to discharge on the seventeenth postoperative day, showed the well functioning graft and heart (Fig. 1D).

CASE II. Aortic sinus aneurysm and aortic regurgitation in a 22 year old asymptomatic man with Marfan's syndrome.³³ A diastolic aortic murmur, discovered during physical examina-

tion prior to blood transfusion, in a tall (6 feet, 5 inches) thin patient with a slight sternal deformity, high arched palate, but normal sized heart (Fig. 2A) led to percutaneous transfemoral retrograde aortography in order to visualize the aorta and aortic sinuses. This revealed aneurysmal dilatation of the aortic sinuses and moderate aortic regurgitation (Fig. 2B).

CASE III. Idiopathic pericardial effusion in a 28 year old man with an unsuspected aneurysm of the ascending aorta, probably due to "forme fruste"³³ type of Marfan's syndrome. This patient was referred from another hospital because of an enlarged cardiac silhouette. Angiocardiograms showed a large pericardial effusion, a normal-sized heart, and a markedly dilated ascending aorta (Fig. 3, A, B and C). Following pericardiocentesis the patient improved and was discharged. He died 3 years later, at the other hospital, of rupture of the aortic aneurysm.

CASE IV. Dextrocardia, situs inversus, single atrium and ventricle with pulmonary arteries to

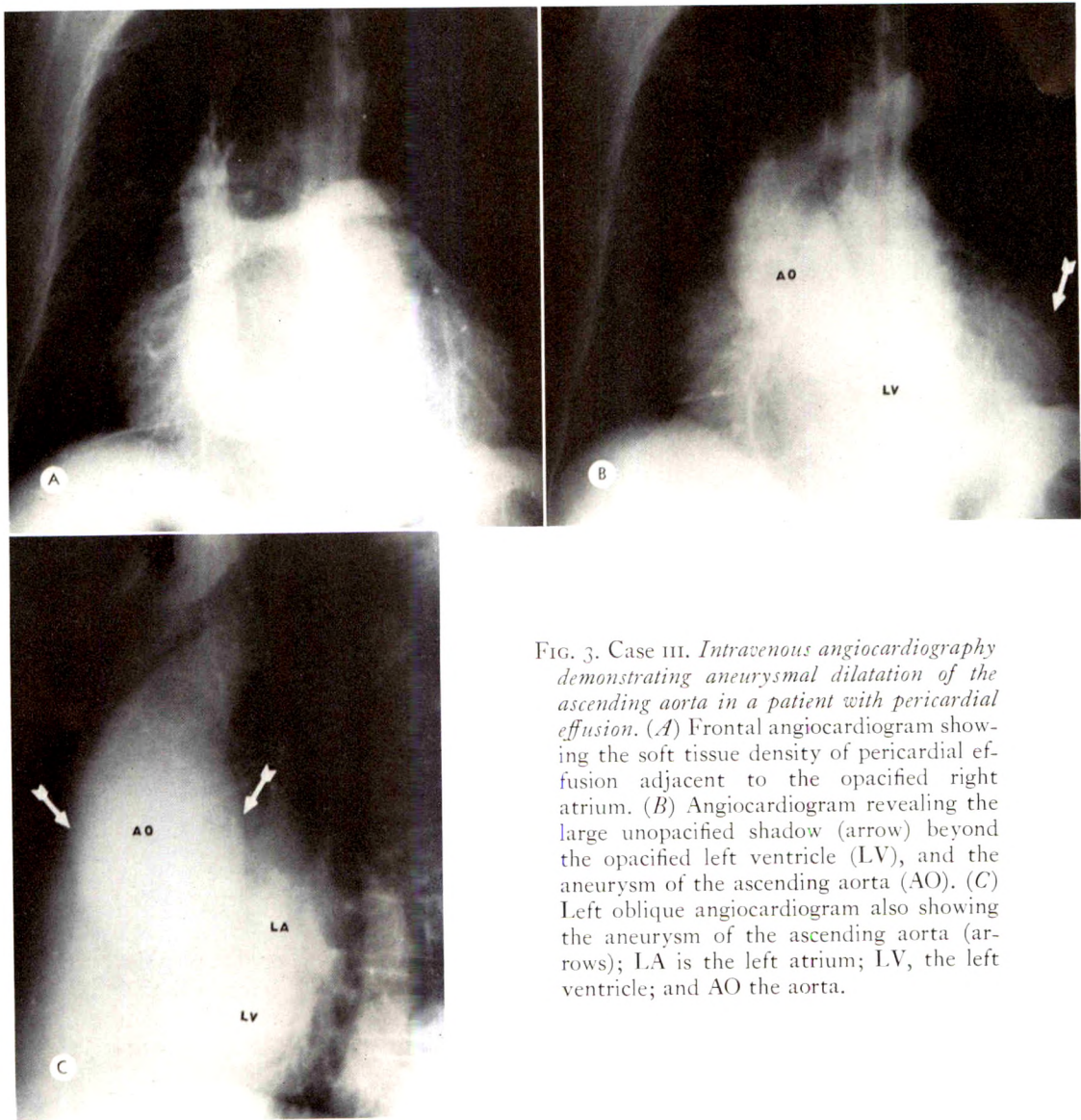


FIG. 3. Case III. *Intravenous angiocardiology demonstrating aneurysmal dilatation of the ascending aorta in a patient with pericardial effusion.* (A) Frontal angiogram showing the soft tissue density of pericardial effusion adjacent to the opacified right atrium. (B) Angiogram revealing the large unopacified shadow (arrow) beyond the opacified left ventricle (LV), and the aneurysm of the ascending aorta (AO). (C) Left oblique angiogram also showing the aneurysm of the ascending aorta (arrows); LA is the left atrium; LV, the left ventricle; and AO the aorta.

upper and lower lobes from truncus arteriosus, anomalous pulmonary veins to single atrium, and bilateral superior venae cavae. A 2 year old female cyanotic child was admitted because of seizures. Examination revealed a thin, cyanotic, polycythemic child with an enlarged heart in the right hemithorax and a plethoric left lung field (Fig. 4A). There was a funnel chest and deformity of the left lower portion of the esophagus (Fig. 4B). A loud continuous murmur was heard over the left hemithorax. Biplane intravenous angiocardiology on September 10, 1963, showed filling of an atrium and ventricle

from which arose a wide aorta. Also arising from the aorta were a pair of pulmonary arteries which inserted into both upper lobes, and below these another set of arteries going into the lower lobe (Fig. 4C). Later in the series, huge pulmonary venous channels were seen connecting with a left superior vena cava and draining into the single atrium (Fig. 4D). Thoracic aortography via the transfemoral route was performed 2 days later in order to better visualize the truncus arteriosus and the anomalous pulmonary arterial and venous systems (Fig. 4, E and F). A later serial angiogram showed retrograde

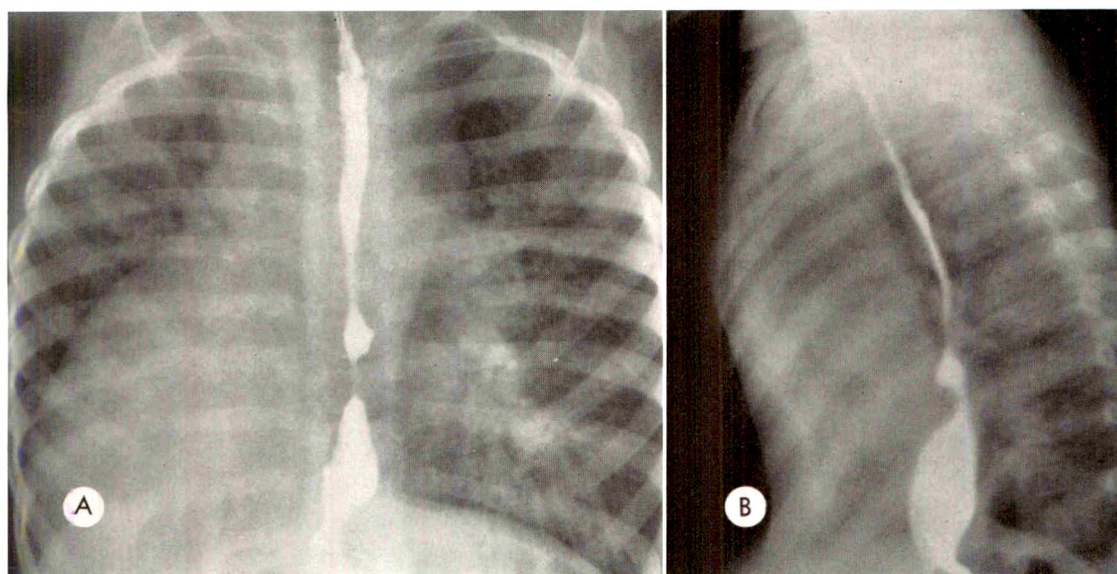


FIG. 4. Case IV. *Truncus arteriosus, pulmonary arteries arising from aorta, anomalous pulmonary veins to single atrium, single ventricle, dextrocardia with situs inversus, and bilateral superior venae cavae demonstrated by intravenous angiocardiology and selective thoracic aortography.* (A) Frontal teleroentgenogram showing a markedly enlarged heart in the right hemithorax and plethoric pulmonary vasculature in left lung. (B) Lateral esophagram demonstrating a funnel chest and indentation of esophagus.

opacification of a left superior vena cava and opacification of the right superior vena cava from the cerebral circulation (Fig. 4G).

CASE V. *Buckling of right subclavian artery¹⁷ in a 60 year old woman.* A pulsatile "lump"

in the right side of the neck in a hypertensive (250/135 mm. Hg) patient was demonstrated by simultaneous bilateral intravenous angiography to be due to a buckled right subclavian artery (Fig. 5). Intravenous abdominal aortography also disclosed a tortuous abdom-

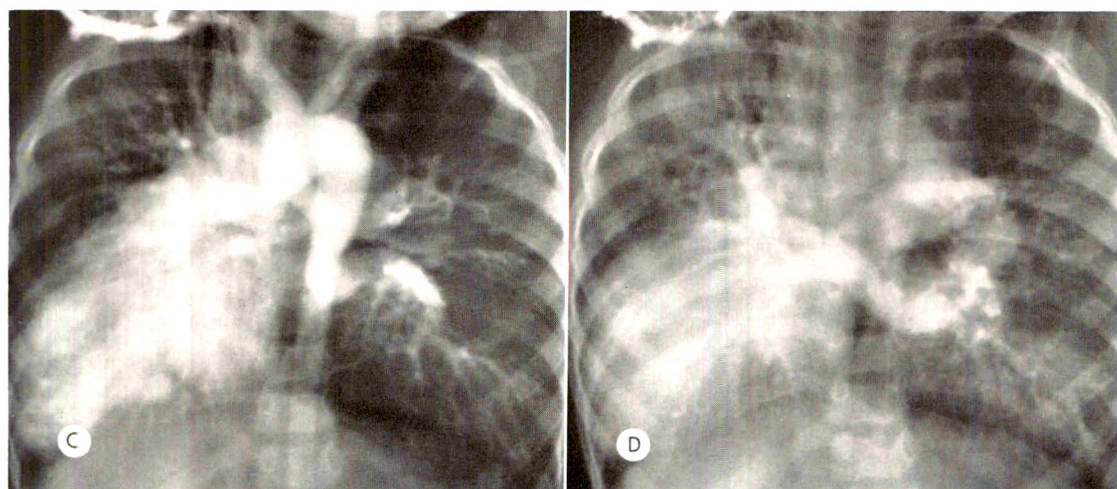


FIG. 4. (C) Frontal angiocardigram revealing a single atrium and ventricle with upper and lower paired pulmonary vessels from the aorta going to the upper and lower lobes of each lung respectively. (D) Later in the series pulmonary veins from both lungs are seen to collect blood in a channel which receives blood from the left vena cava and inserts into the single atrium.

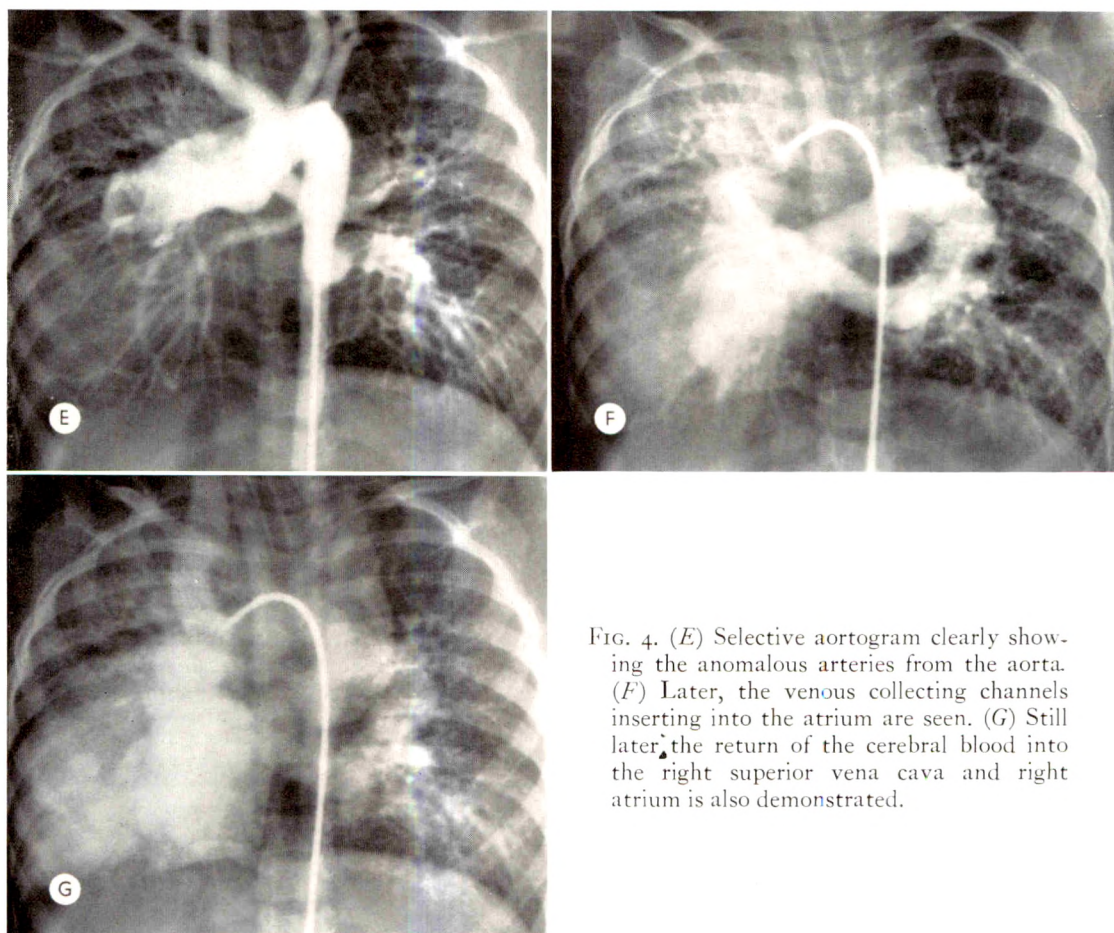


FIG. 4. (E) Selective aortogram clearly showing the anomalous arteries from the aorta. (F) Later, the venous collecting channels inserting into the atrium are seen. (G) Still later, the return of the cerebral blood into the right superior vena cava and right atrium is also demonstrated.

inal aorta, aortoiliac, and iliofemoral arteries. An arteriosclerotic plaque was also seen at the origin of the right renal artery. Percutaneous retrograde transfemoral arteriography in order to better visualize the renal arteries could not be performed because of severe iliofemoral tortuosity.

CASE VI. Buckling of the innominate artery¹³ in a 72 year old hypertensive (180/100 mm. Hg) Negro simulating mediastinal tumor. This patient was admitted to the hospital after conventional roentgenograms of the chest showed a right superior anterior mediastinal mass (Fig. 6, A and B). Brachiocephalic arteriography via the percutaneous transfemoral route demonstrated buckling of the right innominate artery (Fig. 6, C and D).

CASE VII. Aneurysmal dilatation of innominate artery in a 43 year old hypertensive (190/95 mm. Hg) and arteriosclerotic Negroes. A pulsatile

mass in the right side of the neck had been present for 5 years in this patient. Percutaneous transfemoral selective arteriography of the innominate artery disclosed aneurysmal dilatation of the innominate artery with buckling and dilatation of the right carotid artery (Fig. 7, A and B). Surgery was not suggested to the patient because of widespread arteriosclerotic involvement of the peripheral vascular system.

CASE VIII. Coarctation of the aorta in a 49 year old woman. At the age of 35 years, while in Puerto Rico, this patient developed palpitation, chest pain, and hypertension. At the age of 44 years, subtotal gastrectomy for a bleeding peptic ulcer was performed at another hospital. Coarctation of the aorta was also diagnosed but operation was not advised. She was admitted to this hospital because of dizziness. The blood pressures were: right arm, 225/80; left arm, 200/90; and both legs 140/100 mm. Hg. Whereas both wrist pulses and carotid arteries

were vigorous, the lower extremity pulses were diminished. A roentgenogram of the chest demonstrated enlargement of the heart, chiefly of the left ventricle, a deformed aortic knob, and notching of the ribs (Fig. 8A).

Percutaneous transfemoral aortograms showed occlusion of the thoracic aorta (Fig. 8B). Left axillary selective arteriography was then performed but the catheter could not be advanced into the aorta (Fig. 8C). Serial roentgenograms, however, well demonstrated the huge collaterals (Fig. 8, D, E and F). The next day, intravenous angiocardigrams in the erect seated left oblique position demonstrated two sites of coarctation (Fig. 8G).

CASE IX. *Coarctation of the aorta in an asymptomatic 28 year old Negress.* Hypertension (160/90 mm. Hg) was discovered in both arms during the routine pre-employment physical examination of the patient. The blood pressure in the legs was absent. The conventional roentgenogram of the chest showed a normal-sized heart with deformity of the aortic knob and rib notching. Selective right axillary thoracic aortography in the left anterior oblique position revealed coarctation of the thoracic aorta, dilatation of the innominate and left subclavian arteries, poststenotic dilatation of the descending aorta and numerous collaterals (Fig. 9A). Another study in frontal view also showed the coarctation of the aorta and the widespread collaterals (Fig. 9B). Subsequently, the coarctated segment was resected and the aorta reapproximated. Recovery was uneventful and the patient became normotensive.

DISCUSSION

The diagnosis of aortic sinus dilatation and aneurysm of the ascending aorta, which frequently accompanies Marfan's syndrome, can be readily made by intravenous angiocardiology.^{21-24,28,33} The "forme fruste" type of Marfan's syndrome should be suspected when aneurysmal dilatation of the ascending aorta and aortic sinuses is demonstrated without skeletal or eye stigmata (Fig. 1C; and 3, B and C).³³ Aortic insufficiency which is often associated with dilatation of the aortic sinuses can best be demonstrated by selective aortography (Fig. 2B). Indeed, the latter technique is essential for visualizing the



FIG. 5. Case v. Buckling of the right subclavian artery demonstrated with the intravenous method of angiocardiology.

aortic cusps, coronary arteries,^{20,44} aortic sinus perforations, coronary arteriovenous fistulas^{30,37} and aortopulmonary windows.

Intravenous angiocardiology is often satisfactory for the diagnosis of complex congenital heart disease such as truncus arteriosus (Fig. 4, C and D). In such a case, the large pulmonary arteries arising from the aorta and the anomalous veins communicating with a left superior vena cava and draining into a single atrium were recognized. However, this was definitively demonstrated by selective aortography (Fig. 4, E, F and G).

The origins of the brachiocephalic arteries can usually be well visualized with intravenous angiocardiology (Fig. 5).^{13,14} More exquisite details, especially of the bifurcation of the carotid arteries, can be secured with selective opacification of the great arteries (Fig. 6, C and D; and 7, A and B).^{9,10,12,15,38,43} The intravenous tech-

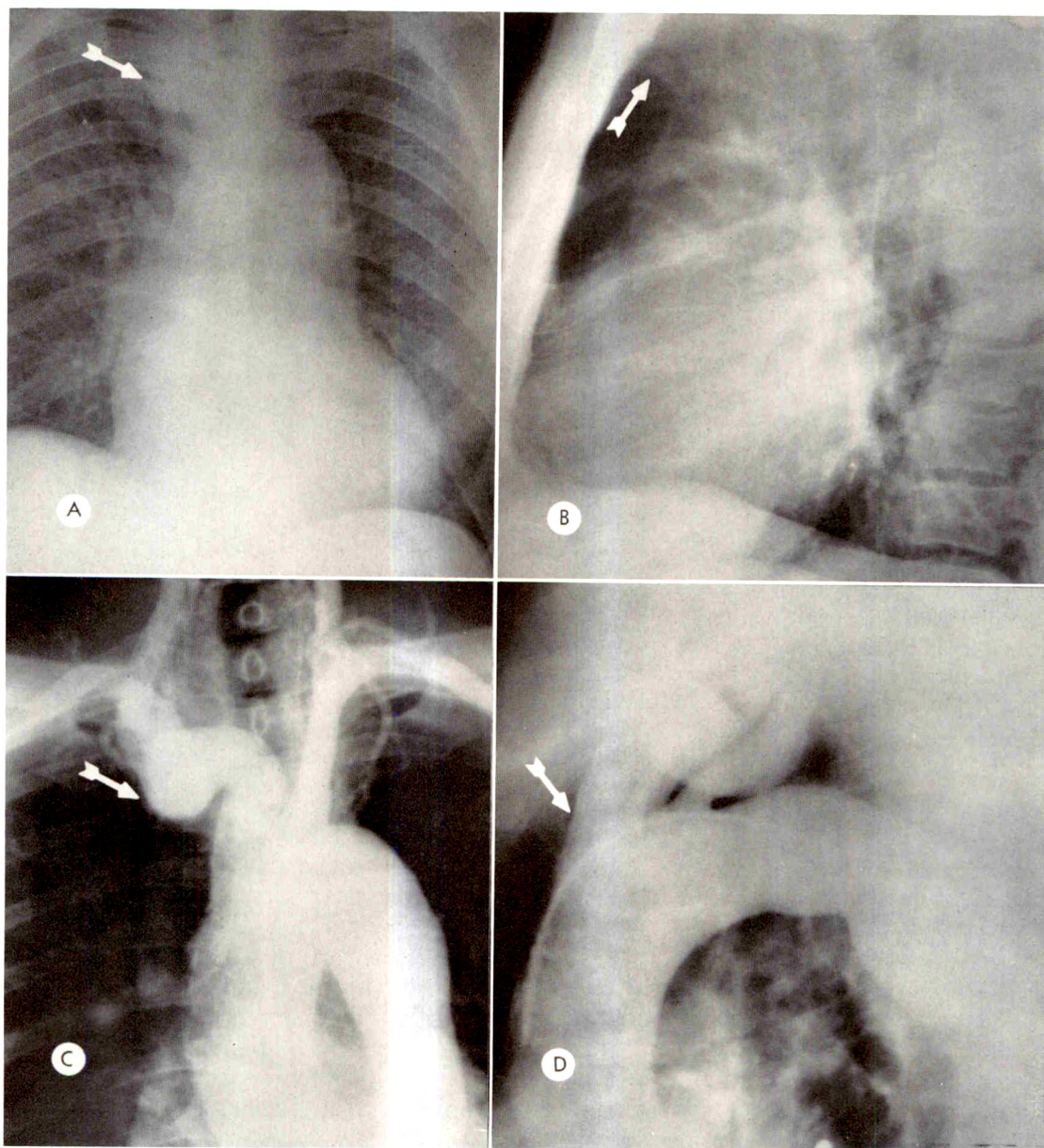


FIG. 6. Case VI. *Buckling of the innominate artery demonstrated by opacification of the great vessels through a catheter in the aortic arch.* (A) Teleroentgenogram showing cardiac enlargement (due to hypertension) and a right superior mediastinal mass (arrow). (B) Left oblique roentgenogram of the chest showing the anterior location of the mass (arrow). (C) Frontal aortogram revealing the buckled right innominate artery (arrow). (D) Oblique aortogram showing the unfolded and dilated innominate artery (arrow).

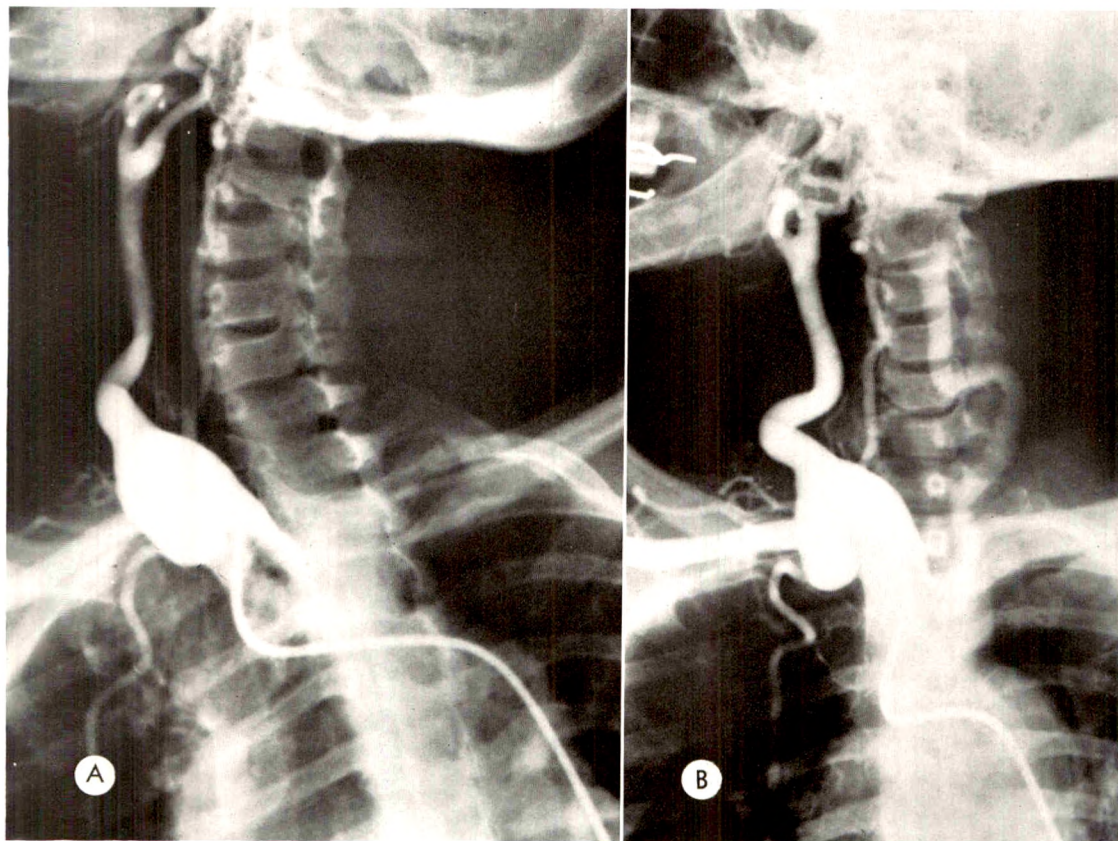


FIG. 7. Case VII. Selective innominate arteriograms showing an aneurysm of the innominate artery. (A) Left oblique view of aneurysmal dilatation of the innominate artery in an arteriosclerotic patient. (B) Frontal view also showing dilatation and tortuosity of the right common carotid artery.

nique has been used for many years to demonstrate coarctation of the aorta (Fig. 8G).^{2,3,8,22,31} Figure 8, B and C, shows that the selective transfemoral and axillary arterial routes may rarely be unsuccessful for visualizing the lesion; however, it does appear that selective aortography is superior to the intravenous technique for the visualization of the collateral circulation in coarctation of the aorta (Fig. 8, D, E and F; and 9, A and B).

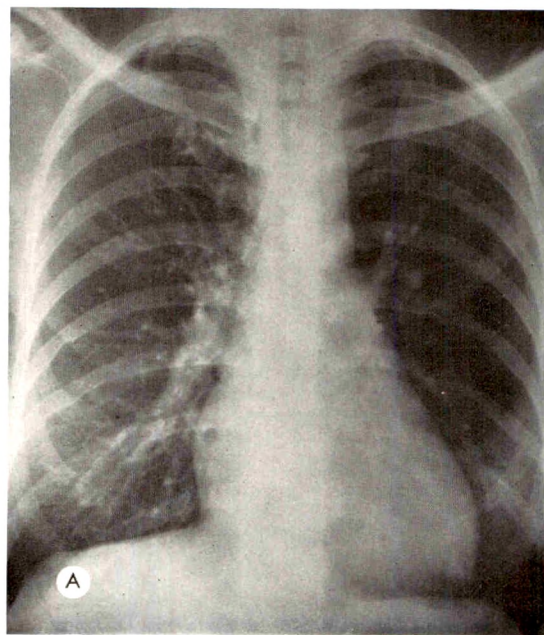


FIG. 8. Case VIII. Intravenous and selective visualization of coarctation of the aorta. (A) Frontal teleroen-genogram showing left ventricular enlargement, deformity of the aortic arch, and rib notching.

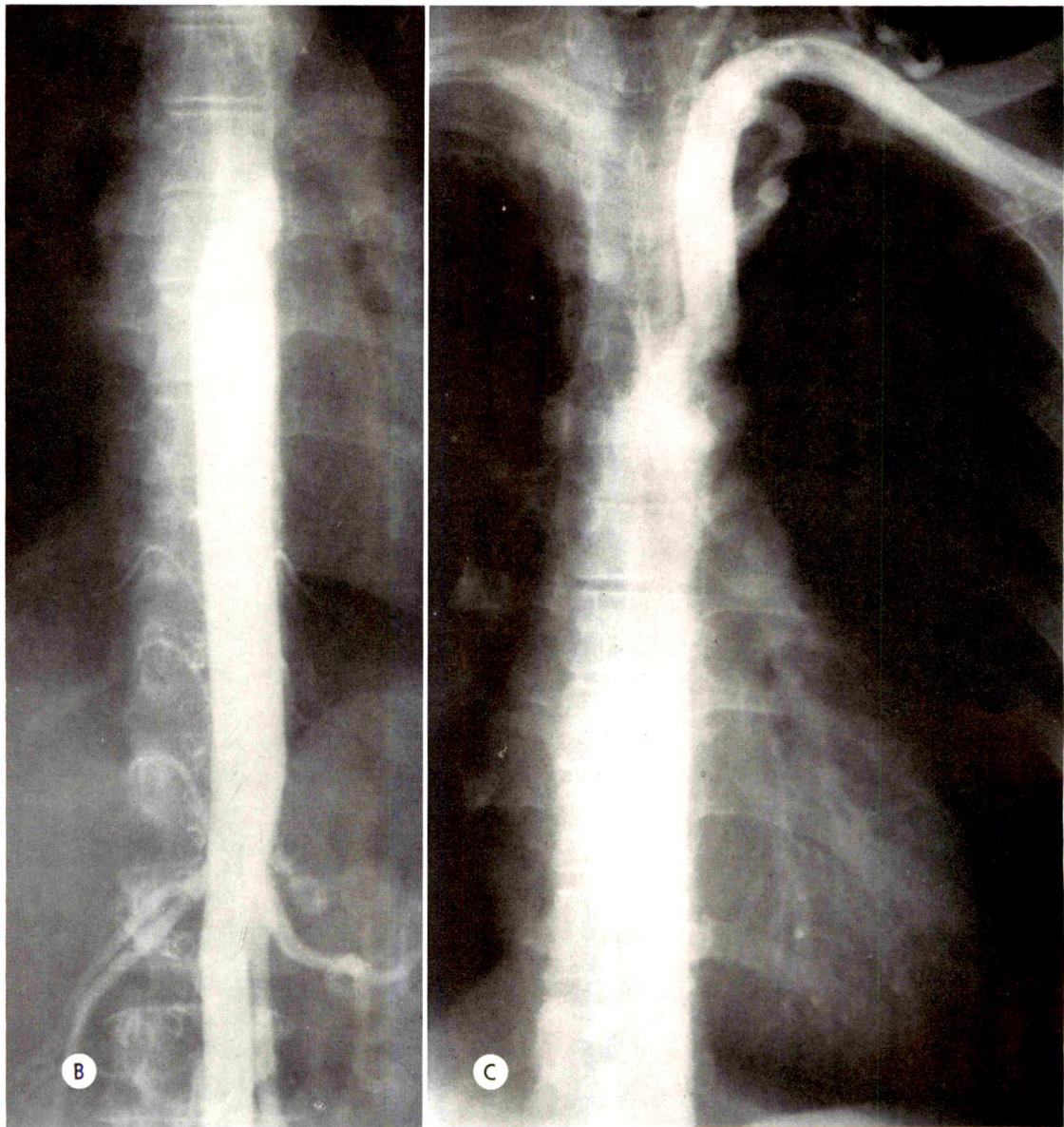


FIG. 8. (B) Percutaneous transfemoral aortogram demonstrating occlusion of the descending thoracic aorta. (C) Left axillary selective arteriogram showing stenosis of the subclavian artery as it joins the aorta.

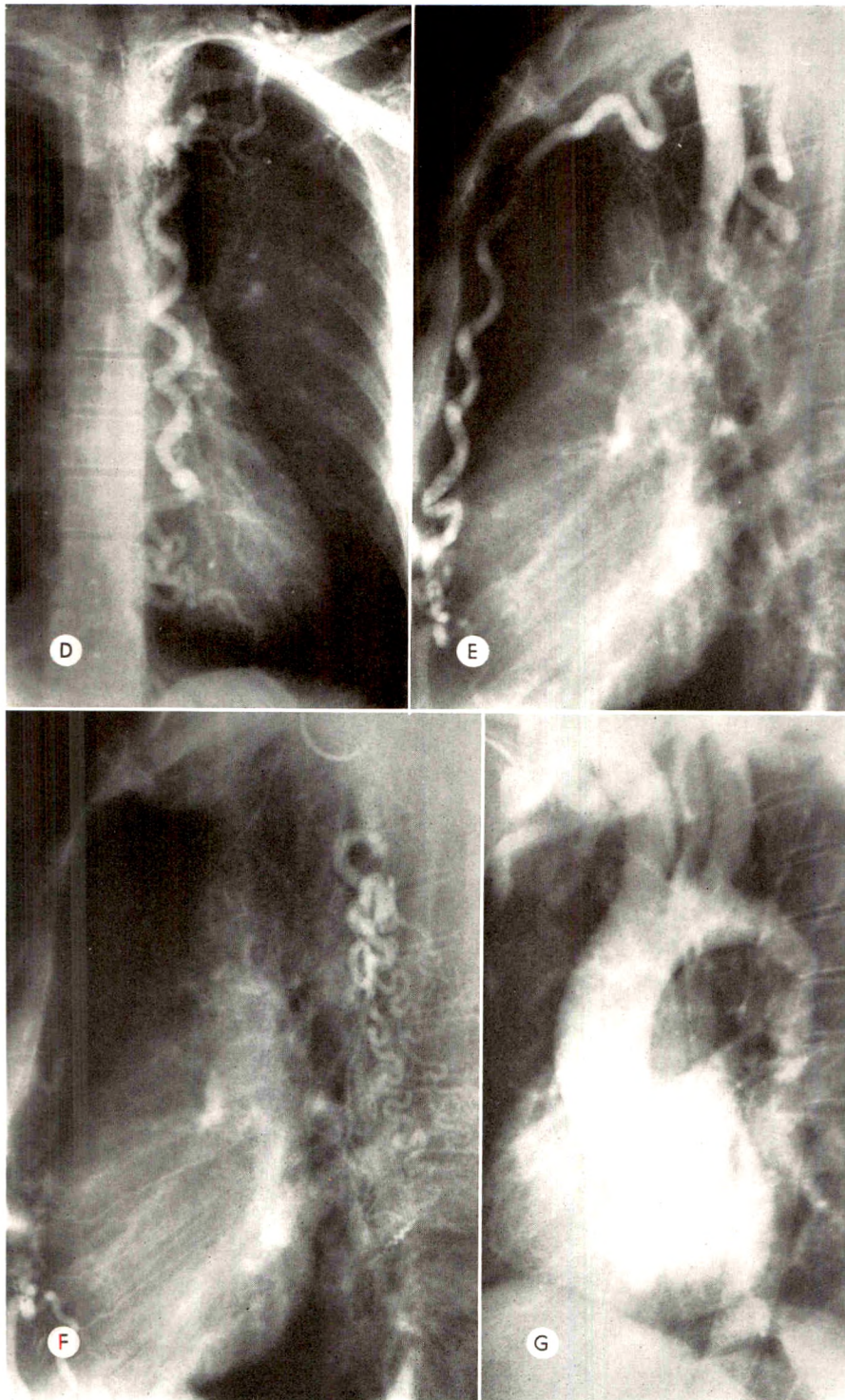


FIG. 8. (D) Frontal selective arteriogram at a later time showing the huge left internal mammary collateral blood flow. (E) Lateral arteriogram of the axillary study, biplane of D, also showing the collateral circulation. (F) At a later time, the exquisite details of the thoracic collaterals are shown. (G) Left oblique angiogram showing the doubly coarctated segments of the aorta.

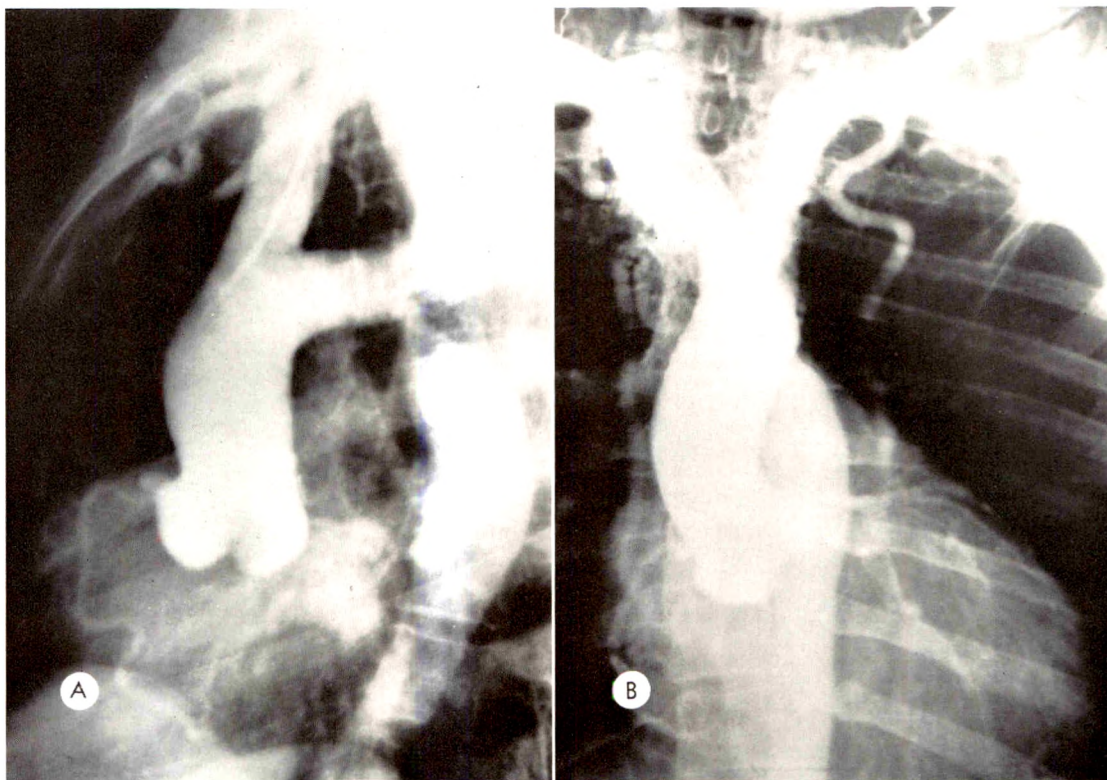


FIG. 9. Case 1X. *Axillary selective arteriography demonstrating coarctation of the aorta. (A) Left oblique aortogram. (B) Frontal aortogram.*

There is general agreement that supra-valvular stenosis of the aorta can best be demonstrated by selective aortography and furthermore this permits diagnostic hemodynamic pressure recordings.^{6,7} Recently, the association of pulmonic stenosis of the valves or branches has been reported.^{1,45} It would seem desirable, therefore, also to routinely visualize the pulmonary arterial system. Figure 10, *A* and *B*, shows how well this can be done by intravenous angiocardiology. While selective angiocardiology can be used for pulmonary arterial visualization (Fig. 11, *A* and *B*), this technique may obscure the details of the supra-valvular aortic lesion (Fig. 11, *C* and *D*). More reliable is the direct supra-valvular aortic opacification technique (Fig. 11, *E* and *F*).

SUMMARY AND CONCLUSIONS

For over 25 years intravenous angiocardiology has proved very successful for

visualizing the thoracic aorta and its major branches. Aortic sinus aneurysms, thoracic aortic aneurysms including dissecting hematomas, coarctation of the aorta, and the brachiocephalic arteries have also been very well demonstrated.

However, the aortic cusps, coronary arterial circulation, aortic sinus ruptures, coronary arteriovenous fistulas, and supra-valvular aortic stenosis require selective opacification in order to reveal the blood flow, unencumbered by the opacified neighboring cardiovascular structures. Although aneurysms and buckling of the brachiocephalic arteries can be demonstrated by intravenous angiocardiology, arteriosclerotic plaques and occlusive disease at the origins of the great vessels and at the carotid bifurcations require selective arteriography of these vessels. Similarly, truncus arteriosus, aortopulmonary window, and patent ductus arteriosus can best be diagnosed by selective aortography techniques.



FIG. 10. *Supravulvar aortic stenosis in a 28 year old man.* (A) Intravenous frontal angiogram showing the intact pulmonary arterial system. (B) Left oblique angiogram also showing pulmonary arteries. (C) Frontal selective aortogram showing the supravulvar aortic stenosis. (D) Lateral aortogram, biplane of C, also showing the supravulvar aortic stenosis.

Coarctation of the aorta can be well demonstrated with intravenous angiography; however, the collateral circulation is more clearly defined with the catheter technique.

Improvements in serial roentgen apparatus have greatly enhanced contrast roent-

genography of the thoracic aorta. Mechanical pressure devices have also improved the injection of contrast agents into the various parts of the cardiovascular system. For the most efficient result in demonstrating a thoracic aortic lesion, the angiographer should be acquainted with both the

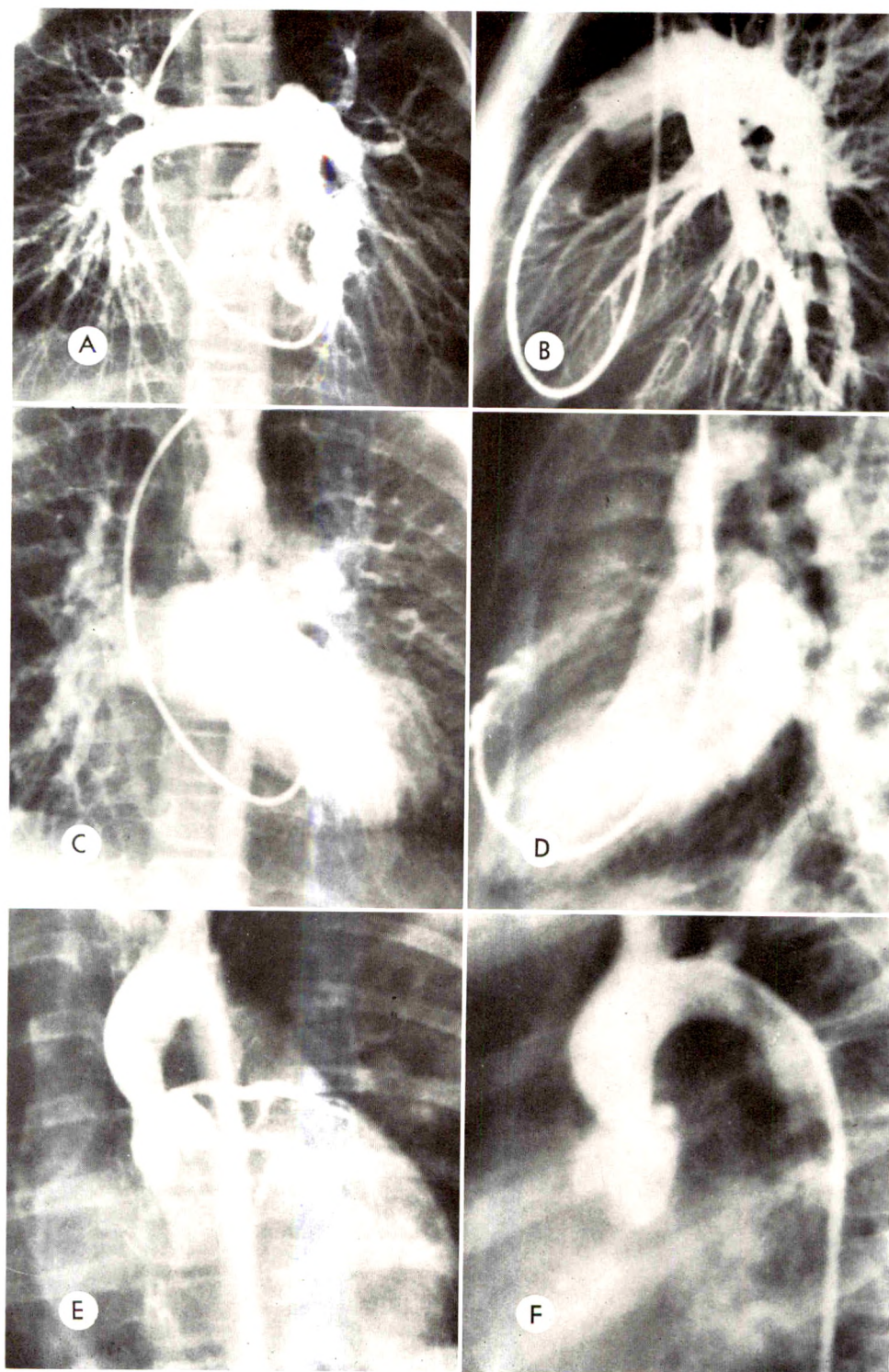


FIG. 11. Supravulvar aortic stenosis in a 4 year old girl showing superiority of selective aortic valvular visualization over selective right ventricular and pulmonary arterial angiography. (A) Frontal selective angiogram with catheter in outflow tract of the right ventricle showing an intact pulmonary arterial system. (B) Biplane of A, showing normal pulmonary arteries. (C) Only the supraortic stenosis but

intravenous and selective techniques. Ideally, he should be in a position to select the procedure which will establish the diagnosis definitively. In some cases it may be necessary, in order to secure more complete information regarding thoracic aortic disease, to perform both techniques.

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COLLATERAL CIRCULATION IN AORTO-ILIO-FEMORAL OCCLUSIVE DISEASE*

AS DEMONSTRATED BY A UNILATERAL PERCUTANEOUS COMMON FEMORAL ARTERY NEEDLE INJECTION

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ANGIOGRAPHIC examination of the abdominal aorta and arteries of the pelvis and both lower extremities may be accomplished easily and safely by a manual injection of contrast material directly through a unilateral percutaneous common femoral artery needle.⁵ The purpose of the authors is to emphasize further the value of this technique for aorto-ilio-femoral arteriography and to summarize the collateral circulation in peripheral arterial occlusive disease, especially as demonstrated by this method of examination.

TECHNIQUE

This angiographic technique has evolved from the work of Castellanos and Pereiras,² Fariñas,⁴ Sulamaa,²² Sammons and Mahin,¹⁷ and Schobinger.²⁰ It has been utilized for angiographic examination of pelvic organs^{11,18} and evaluation of occlusive arterial disease.^{17,19}

Under local anesthesia a common femoral artery is cannulated percutaneously with a 205 Seldinger or a Becton-Dickenson 15 gauge thin wall arterial puncture needle, and the needle is advanced under control a few centimeters up the lumen of the vessel. A manual injection of 50 ml. of 60 per cent methylglucamine diatrizoate* or meglumine iothalamate** is made during maximum decrease in intra-aortic pressure which results from sudden release of a Valsalva effort (Fig. 1). During the subsequent rebound rise in pressure, the contrast material flows into the peripheral arteries in a compact bolus. The RIL-400

roentgenographic table* is used which automatically changes four 14×51 inch cassettes, permitting simultaneous roentgenography of the abdomen, pelvis, and lower extremities. Timing of the four exposures varies with the pathology in question. Usually, the first film is exposed immediately before the end of the injection, and subsequent films are exposed at 3 to 5 second intervals. Opacification of the vessels in the extremity opposite the injection is not increased significantly by inflation of a blood pressure cuff to above systolic at the high thigh level distal to the needle. Optimum results are achieved by precise timing of the injection to coincide with the precipitous drop in intra-aortic pressure which follows sudden release of the Valsalva effort.

MATERIAL

The roentgenograms were reviewed of 284 consecutive patients who had bilateral aorto-ilio-femoral arteriography by the above technique for evaluation of occlusive vascular disease. There were 212 men and 72 women; 267 patients were Caucasian and 17 were Negro; 62 patients were diabetic, 186 were not, and this information was not available in 36. Table 1 shows the ages of the patients according to decade.

Two hundred and fifty-five patients had generalized arteriosclerotic disease with arterial occlusions of varying degree. The arteriosclerosis was considered minimal in 75, moderate in 105, and severe in 75 patients. Fifteen patients had arterial occlusion or aneurysm without generalized

* Renografin, E. R. Squibb & Sons, 745 Fifth Ave., New York 22, New York.

** Conray, Mallinckrodt Chemical Works, St. Louis, Missouri.

* Research Instrumentation, Limited, Montreal, Quebec, Canada.

* From The Edward Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Missouri.

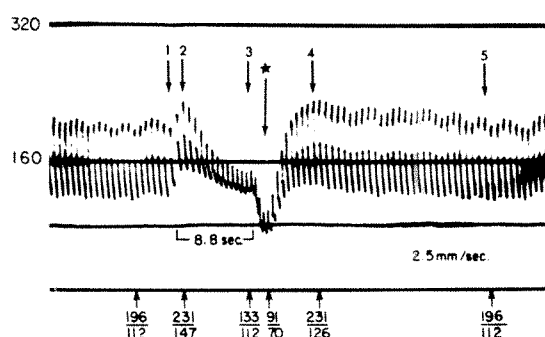


FIG. 1. Intra-aortic pressure recording during performance of the Valsalva maneuver by a hypertensive 36 year old woman with chronic pyelonephritis. The catheter was placed in the distal abdominal aorta percutaneously from the femoral artery. At the onset of forceful exhalation against the closed glottis (arrow 1), there is a slight rise in the intra-aortic pressure (arrow 2). This is followed by a progressive decrease in pressure (arrows 2-3) which does not reach its maximum for at least 8 or 10 seconds (arrow 3). When the patient suddenly releases the Valsalva effort (arrow 3), there is a precipitous additional decrease in intra-aortic pressure. The injection of contrast material is made at this precise moment (arrow with star) during maximum decrease in pressure. In this patient blood pressure decreased from 196/112 to 91/70 mm. Hg. During the subsequent rebound rise in pressure (arrow 4), contrast material flows in a compact bolus into peripheral arteries. Pressure then gradually returns to resting levels over a period of several seconds (arrow 5).

arteriosclerosis, and in 14 patients the examination was normal. Table II shows the number of complete or partial segmental occlusions, aneurysms, or acute emboli, demonstrated in the aorta or major arteries of the pelvis or thighs. Thus, there was available for study the collateral circulation around 263 complete and 199 partial arterial occlusions in a total of 270 patients having abnormal aorto-ilio-femoral arteriograms.

VISCERAL ARTERY COLLATERALS

Anastomoses may develop between the 3 major components of the visceral arterial system in the abdomen and pelvis, *i.e.*, the superior mesenteric artery, the inferior mesenteric artery, and the visceral branches of the hypogastric arteries.

TABLE I

AGE DISTRIBUTION OF 284 PATIENTS HAVING BILATERAL AORTO-ILIO-FEMORAL ARTERIOGRAPHY VIA A UNILATERAL COMMON FEMORAL ARTERY

NEEDLE INJECTION

Age in Years	No. of Patients
20-29	2
30-39	7
40-49	34
50-59	106
60-69	87
70-79	41
80-89	7

Figure 2 is a composite line drawing showing schematically the major visceral pathways of collateral circulation which have been demonstrated.

In the presence of occlusive disease of the abdominal aorta, the *superior and inferior mesenteric* arteries are the visceral artery sources of collateral circulation to the pelvis. The most proximal portion of a complete occlusion of the abdominal aorta almost invariably is immediately distal to the origin of either the renal or the inferior mesenteric artery. When a complete aortic occlusion is limited entirely to the area *between* the origins of the renal and inferior mesenteric arteries, then the middle colic branch of the superior mesenteric artery bypasses the aortic occlusion by anastomosing with the left colic branch of the inferior mesenteric artery. Retrograde flow is thus established in the inferior mesenteric artery which empties into the abdominal aorta distal to the occlusion (Fig. 3, *A* and *B*).

When the aortic occlusion begins immediately distal to the origin of the renal arteries and *includes* the origin of the inferior mesenteric artery, then blood flow also is from the middle colic branch of the superior mesenteric artery to the left colic branch of the inferior mesenteric artery. However, the left colic artery then supplies the superior hemorrhoidal branches of the inferior mesenteric artery which anastomose with the middle hemorrhoidal

TABLE II
DISTRIBUTION OF SEGMENTAL OCCLUSIVE DISEASE IN 270 PATIENTS
WITH ABNORMAL AORTO-ILIO-FEMORAL ARTERIOGRAMS

		Complete Segmental Occlusions	Partial Segmental Occlusions	Aneurysms	Acute Emboli
Aorta		5*	6	12	—
Common iliac	—right	16	27	12	6
	—left	28	29	12	1
External iliac	—right	10	9	—	2
	—left	7	13	2	1
Hypogastric	—right	10	16	—	—
	—left	18	24	2	—
Common femoral	—right	7	4	2	—
	—left	9	12	2	1
Deep femoral	—right	3	6	—	2
	—left	6	6	—	4
Superficial femoral	—right	76	24	1	2
	—left	68	23	2	1
Total number of lesions		263	199	47	20

* Including 3 patients with Leriche syndrome.

branches of the hypogastric arteries and the inferior hemorrhoidal branches of the internal pudendal arteries.

When a complete aortic occlusion is located entirely *distal* to the origin of the inferior mesenteric artery, the block is bypassed by the superior hemorrhoidal branch of the inferior mesenteric artery which anastomoses with one or both hypogastric arteries via the above-mentioned hemorrhoidal collateral pathways (Fig. 4). This collateral channel also may be demonstrated in patients with common iliac (Fig. 5, *A* and *B*) and/or other major pelvic artery occlusions.

These visceral collateral channels have been documented by others in several case reports.^{1,10,16} At times they are extremely large and serpiginous. They may be demonstrated by retrograde catheter or translumbar aortography, but also may be shown, at least in part, by the technique of

aorto-ilio-femoral arteriography described here.

We have examined 1 patient with fibromuscular hyperplasia involving the superior mesenteric artery proximal to the origin of its middle colic branch. Blood flow to the superior mesenteric artery was via its middle colic branch from the left colic branch of the inferior mesenteric artery. This particular collateral channel has been reported thus far only in patients with congenital occlusive disease of the proximal superior mesenteric artery.¹⁴

In the pelvis the visceral artery collaterals arise from the *vesical*, *middle hemorrhoidal* and in females the *uterine* and *vaginal* branches of the anterior trunks of the hypogastric arteries and also from the *inferior hemorrhoidal* branches of the internal pudendal arteries. These branches often contribute to a rich network of transpelvic

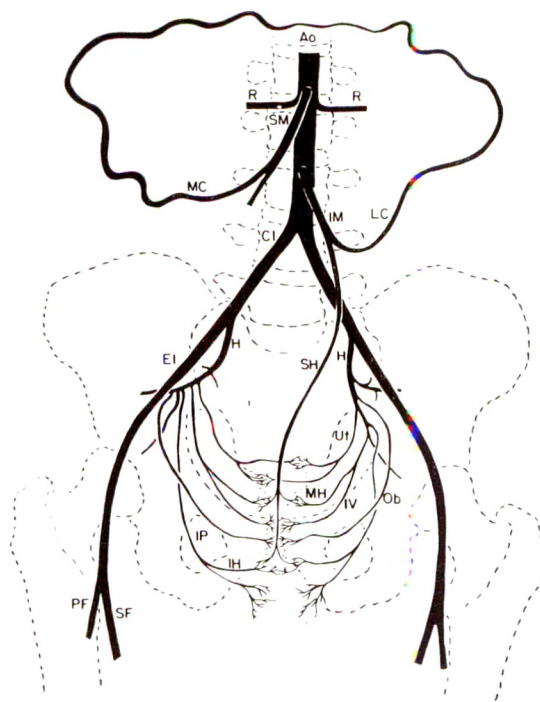


FIG. 2. Composite line drawing showing schematically the major visceral pathways of collateral circulation demonstrated in aorto-ilio-femoral occlusive disease.

Ao	aorta	MC	middle colic
CI	common iliac	MH	middle hemorrhoidal
EI	external iliac	Ob	obturator
H	hypogastric	PF	profunda femoris
IH	inferior hemorrhoidal	R	renal
IM	inferior mesenteric	SF	superficial femoral
IP	internal pudendal	SH	superior hemorrhoidal
IV	inferior vesical	SM	superior mesenteric
LC	left colic	Ut	uterine

collateral vessels which serve to circumvent occlusions in the aorta, common iliac, or hypogastric arteries.

PARIETAL ARTERY COLLATERALS

Parietal arteries may form an extensive network of collateral vessels to circum-

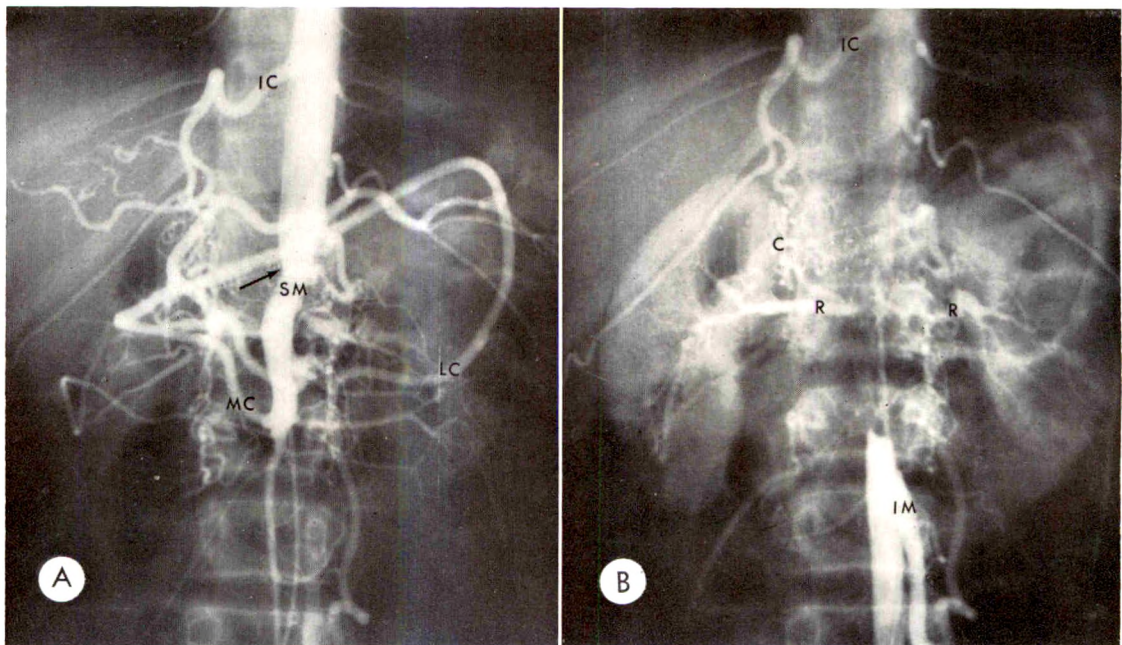


FIG. 3. (A and B) Percutaneous transfemoral retrograde catheter aortogram in an 11 year old girl with high grade congenital stenosis of the abdominal aorta from L1 (arrow) to L3. Blood flow is from the middle colic branch (MC) of an enlarged superior mesenteric artery (SM) to the left colic branch (LC) of the inferior mesenteric artery (IM) which then empties into the distal abdominal aorta by retrograde flow in the inferior mesenteric artery. Blood flow through the renal arteries (R) is compromised severely. There is an enlarged intercostal artery (IC) on the right supplying collateral flow (C) to the right kidney. (Reproduced with permission from *Annals of Surgery*.²¹)

vent an occlusion in any portion of the aorto-ilio-femoral arterial system. Figure 6 is a composite line drawing showing schematically the major parietal pathways of collateral circulation which have been demonstrated.

These collaterals tend to develop principally on the same side of the body as the occlusions they bypass. The most significant parietal collaterals arise from the intercostal, subcostal, lumbar, and middle sacral branches of the aorta; the ilio-lumbar, lateral sacral, superior gluteal, and obturator branches of the hypogastric arteries; the deep iliac circumflex branches of the external iliac arteries; and the medial and lateral circumflex branches of the profunda femoris arteries. The following are the major parietal artery collaterals which have been shown:

FROM THE AORTA

Intercostal and/or subcostal arteries frequently anastomose with the deep iliac circumflex artery (Fig. 5, A and B) and may become quite large. Prominent intercostal and subcostal collateral vessels may produce notching of the lower ribs in the presence of severe occlusive disease of the upper abdominal aorta, but we have not yet observed this phenomenon with occlusive disease of the distal abdominal aorta. Intercostal and/or subcostal arteries also may anastomose with the ilio-lumbar and/or superior gluteal arteries. They may supply the superficial iliac circumflex branch of the common femoral artery, but only rarely was this demonstrated to be a significant collateral pathway (Fig. 7, A and B). *Lumbar* arteries often anastomose with the deep iliac circumflex (Fig. 8), ilio-lumbar (Fig. 8; and 9, A, B and C) or superior gluteal (Fig. 10, A, B and C) arteries. The *middle sacral* branch of the aorta may join ilio-lumbar or lateral sacral (Fig. 9, A, B and C) arteries.

FROM THE HYPOGASTRIC ARTERY

The posterior and anterior trunks of the hypogastric arteries each give rise to 3

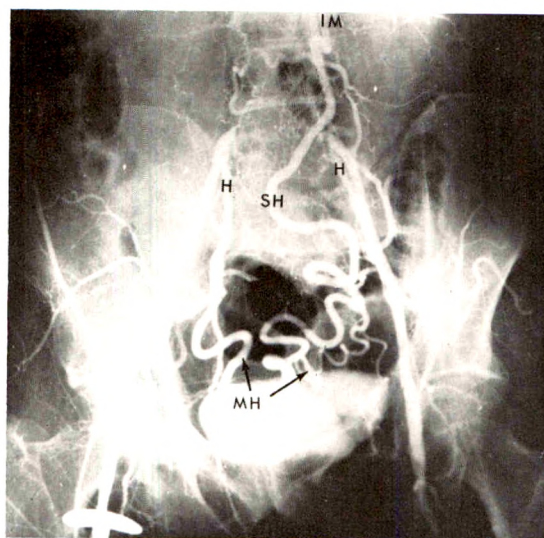


Fig. 4. Manual injection of contrast material through a percutaneous needle in the right common femoral artery in a 60 year old man with the Leriche syndrome. Collateral circulation is from the inferior mesenteric artery (IM) to the hypogastric arteries (H) via extremely undulant and enlarged superior hemorrhoidal (SH) and middle hemorrhoidal (MH) anastomotic channels.

parietal branches which participate significantly in pelvic collateral circulation.

From the posterior trunk. The *ilio-lumbar* artery may anastomose with the deep iliac circumflex on the same side and also may anastomose with the opposite ilio-lumbar artery (Fig. 9, A, B and C). The *lateral sacral* branches of the hypogastric arteries frequently join with each other (Fig. 5, A and B; 8; and 9, A, B and C) and also with the middle sacral branch of the aorta (Fig. 8; and 9, A, B and C). The *superior gluteal* artery may join the deep iliac circumflex on the same side and also may anastomose with the lateral (Fig. 11) or medial femoral circumflex arteries.

From the anterior trunk. The *inferior gluteal* artery may anastomose with the medial femoral circumflex on the same side. The *obturator* artery may anastomose with the medial femoral circumflex (Fig. 9, A, B and C) and with the opposite obturator which, in turn, may join the medial femoral circumflex on its side. The *internal pudendal* arteries may anastomose with each other

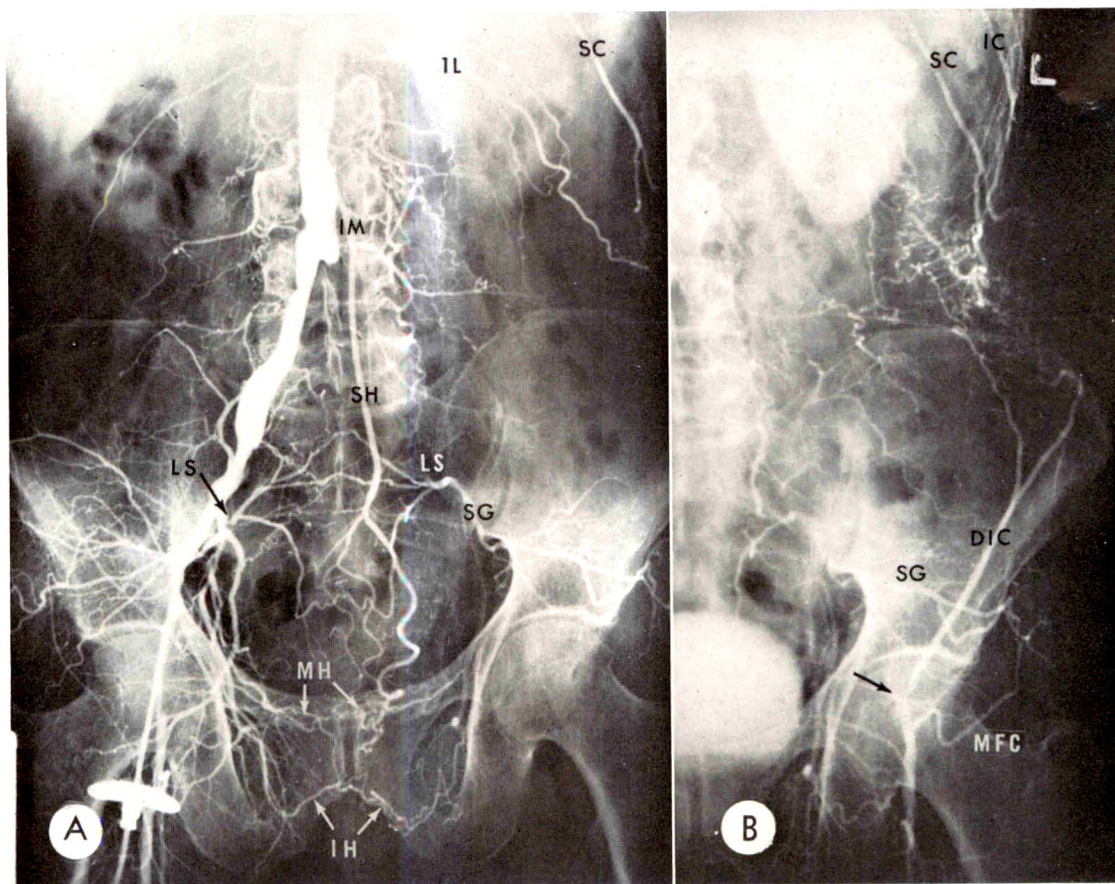


FIG. 5. (A and B) Manual injection of contrast material through a percutaneous needle in the right common femoral artery in a 57 year old man with marked generalized arteriosclerosis and complete occlusion of the left common iliac, hypogastric, and external iliac arteries. The superior hemorrhoidal branches (SH) of the inferior mesenteric artery (IM) are prominent and form collateral channels with the middle hemorrhoidal branches (MH) of the hypogastric arteries and inferior hemorrhoidal branches (IH) of the internal pudendal arteries. The obstructions are further circumvented by prominent transpelvic lateral sacral collaterals (LS) to the distal left hypogastric artery. The left hypogastric artery, in turn, opacifies the superior gluteal (SG) which anastomoses with the medial femoral circumflex (MFC) to help circumvent the occlusions. Enlarged left intercostal (IC), subcostal (SC), and first lumbar (1L) arteries opacify the deep iliac circumflex artery (DIC) which is a major source of supply to the common femoral artery (arrow).

via their perineal and various genital branches. They also may join with the superficial and/or deep external pudendal branches of the common femoral artery or medial femoral circumflex branch of the profunda femoris artery.

FROM THE EXTERNAL ILIAC ARTERY

The *deep iliac circumflex* branch of the external iliac artery may anastomose with the ilio-lumbar (Fig. 9, A, B and C; and 12, A and B) and/or superior gluteal arteries on the same side. The *inferior epigastric*

branch of the external iliac artery may be a significant collateral channel, receiving blood from the superior epigastric branch of the internal mammary artery (Fig. 13, A, B and C).

FROM THE COMMON FEMORAL ARTERY

The *superficial iliac circumflex* branch of the common femoral artery occasionally is a significant collateral vessel, receiving blood from subcostal, intercostal, or lumbar arteries (Fig. 7, A and B). The *external pudendal* branches of the common femoral ar-

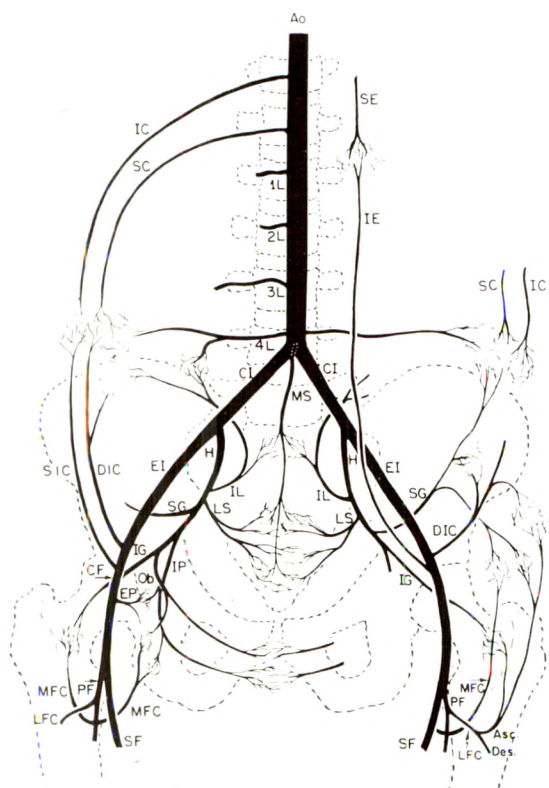


FIG. 6. Composite line drawing showing schematically the major parietal pathways of collateral circulation demonstrated in aorto-ilio-femoral occlusive disease.

Ao	aorta	LFC	lateral femoral circumflex
CF	common femoral	Asc.	ascending branch
CI	common iliac	Des.	descending branch
DIC	deep iliac circumflex	LS	lateral sacral
EI	external iliac	MFC	medial femoral circumflex
EP	external pudendal	MS	middle sacral
H	hypogastric	Ob	obturator
IC	intercostal	PF	profunda femoris
IE	inferior epigastric	SC	subcostal
IG	inferior gluteal	SE	superior epigastric
IL	ilio-lumbar	SF	superficial femoral
IP	internal pudendal	SG	superior gluteal
L	lumbar	SIC	superficial iliac circumflex

tery may anastomose with the internal pudendal artery.

FROM THE PROFUNDA FEMORIS ARTERY

The *medial femoral circumflex* branch of

the profunda femoris artery may receive blood from the obturator (Fig. 9, *A*, *B* and *C*), inferior gluteal, or lateral femoral circumflex arteries. The *lateral femoral circumflex* branch may anastomose with the superior gluteal (Fig. 11), inferior gluteal, deep iliac circumflex, or medial femoral circumflex arteries.

In the presence of occlusive disease, involving the superficial femoral artery, the profunda femoris is the only significant source of collateral circulation to the lower extremity. An abundant network of collateral vessels arising from *perforator* and *muscular* branches of the profunda femoris frequently extends distally to reconstitute the popliteal artery. One of the main collateral channels from the profunda femoris to the popliteal is the descending branch of the lateral femoral circumflex artery which anastomoses with the superior lateral genicular artery near the adductor exit.

DISCUSSION

Angiography of the aorto-ilio-femoral arterial system in patients with occlusive vascular disease may be accomplished by means of translumbar aortography, percutaneous transfemoral retrograde catheter aortography, right atrial aortography, or retrograde left brachial aortography with or without a catheter. The difficulties and hazards of translumbar aortography are well documented.¹³ The other techniques also incur unnecessary risks or fail to provide sufficiently intense opacification of the involved vessels for their complete evaluation.

On the other hand, detailed examination of the aorto-ilio-femoral arterial systems bilaterally can be accomplished with essentially no risk to the patient by a manual injection of contrast material directly through a unilateral percutaneous common femoral artery needle. The technique is as easily performed and as well tolerated as routine femoral arteriography, but it provides considerably more information. The complications that have occurred in 284 examinations have been infrequent and minor. One

patient developed transient hypotension during cannulation of the common femoral artery and before injection of contrast material. This was immediately corrected by 0.5 ml. of intravenous methoxamine hydrochloride,* and the examination was completed successfully. In another patient extravasation of contrast material occurred in the region of the needle puncture, because the needle was not advanced sufficiently far up the lumen of the artery prior to injection. The needle was repositioned, and the examination was completed successfully. Occasionally, a clinically insignificant hematoma developed at the puncture site,

* Vasoxyl, Burroughs Wellcome and Co., Inc., 1 Scarsdale Rd., Tuckahoe 7, New York.

immediately controlled by manual compression.

Descriptions of the normal anatomy and collateral circulation of the aorto-ilio-femoral arterial system thus far have been based on autopsy⁶ and surgical¹⁵ experience, as well as femoral arteriography^{7,9,12} and translumbar aortography.^{1,3,10,14,16,23} Bilateral aorto-ilio-femoral arteriography by the technique described has revealed pathways of collateral circulation essentially identical to those described by other methods of examination. It has demonstrated a dynamic relationship between many arteries of the thorax, abdomen, pelvis, and lower extremities. These regions are linked potentially by a rich network of visceral and/or

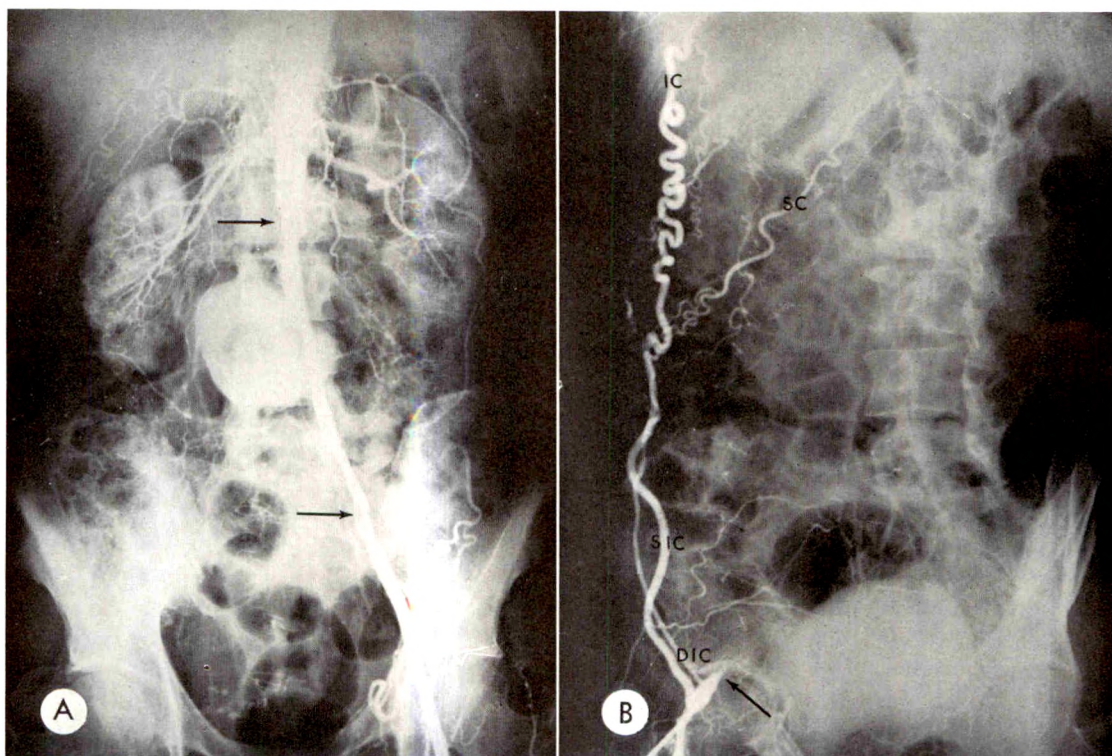


FIG. 7. (A) Manual injection of contrast material through a percutaneous needle in the left common femoral artery in a 52 year old man who had a distal aorto-bilateral common iliac graft (arrows) placed 7 years previously. He was asymptomatic, but physical examination revealed a pulsatile sensation in the region of the aortic bifurcation, indicating probable aneurysmal dilatation of the graft. The aortic and left common iliac portions of the graft are normal, but a large aneurysm has developed in the right common iliac segment with complete obstruction distally. (B) Manual injection of contrast material through a percutaneous needle in the right common femoral artery with the patient in a right posterior oblique position. The distal extent of the obstruction (arrow) at the origin of the common femoral artery is demonstrated. There are collateral channels between intercostal (IC) and subcostal (SC) arteries and the deep iliac circumflex (DIC) and enlarged superficial iliac circumflex (SIC) arteries.

parietal collateral vessels which may bypass occlusions in any segment of the aorto-ilio-femoral arterial system. Connections between the visceral and parietal systems may develop, the principal links being hypogastric branches, some of which are visceral and some parietal. With occlusion of a major artery in the aorto-ilio-femoral system, the collateral vessels which develop follow the described pathways in more or less predictable patterns.

SUMMARY

A safe and effective technique of bilateral aorto-ilio-femoral arteriography by a manual injection of contrast material through a unilateral percutaneous common femoral artery needle is described briefly. The contrast material is injected during the precipitous drop in intra-aortic pressure which follows sudden release of the Valsalva effort. The technique is considered the method of choice for angiographic evaluation of occlusive vascular disease involving the abdominal aorta and arteries of the pelvis and both lower extremities. The collateral circulation in peripheral arterial occlusive disease, especially as demonstrated by this method of examination, is summarized.

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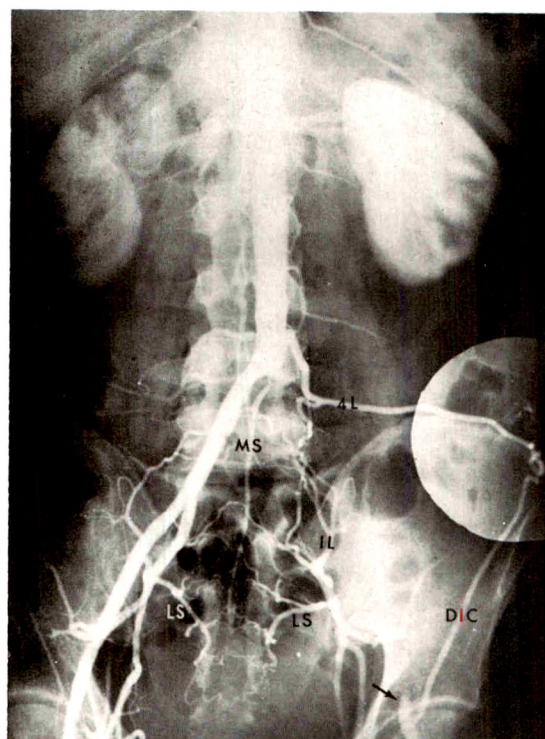


FIG. 8. Manual injection of contrast material through a percutaneous needle in the right common femoral artery in a 43 year old man who suddenly developed left lower extremity pain $1\frac{1}{2}$ years previously and had intermittent claudication since. There is complete occlusion of the left common iliac, hypogastric, and external iliac arteries. A prominent left fourth lumbar artery (4L) anastomoses with the deep iliac circumflex artery (DIC) to supply the left lower extremity (arrow). Transpelvic lateral sacral collaterals (LS) opacify the branches of the left hypogastric artery, aided by an anastomosis between a branch of the left fourth lumbar (4L) and ilio-lumbar (IL) arteries. The middle sacral (MS) contributes to collateral circulation by joining lateral sacral (LS) arteries.

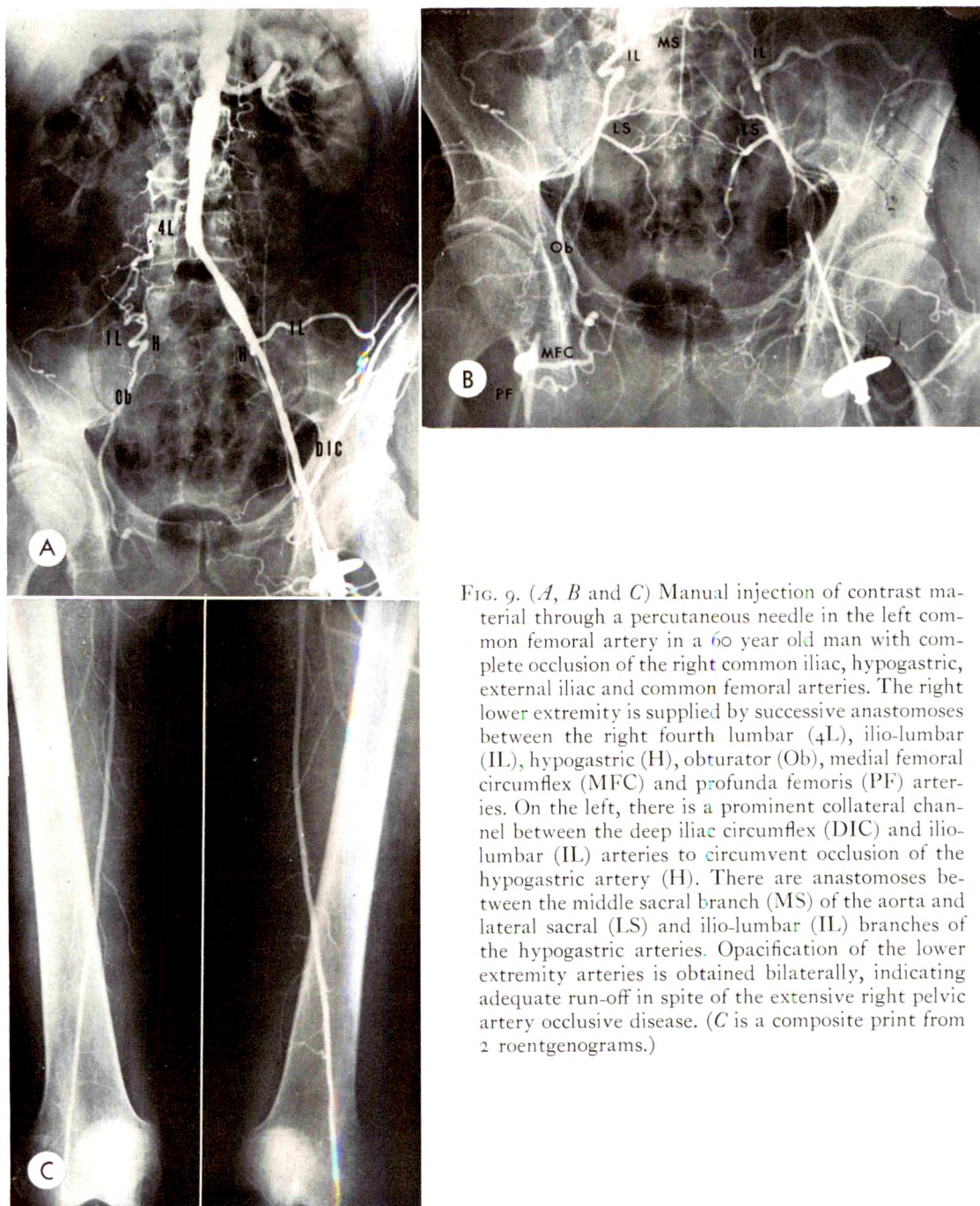


FIG. 9. (A, B and C) Manual injection of contrast material through a percutaneous needle in the left common femoral artery in a 60 year old man with complete occlusion of the right common iliac, hypogastric, external iliac and common femoral arteries. The right lower extremity is supplied by successive anastomoses between the right fourth lumbar (4L), ilio-lumbar (IL), hypogastric (H), obturator (Ob), medial femoral circumflex (MFC) and profunda femoris (PF) arteries. On the left, there is a prominent collateral channel between the deep iliac circumflex (DIC) and ilio-lumbar (IL) arteries to circumvent occlusion of the hypogastric artery (H). There are anastomoses between the middle sacral branch (MS) of the aorta and lateral sacral (LS) and ilio-lumbar (IL) branches of the hypogastric arteries. Opacification of the lower extremity arteries is obtained bilaterally, indicating adequate run-off in spite of the extensive right pelvic artery occlusive disease. (C is a composite print from 2 roentgenograms.)

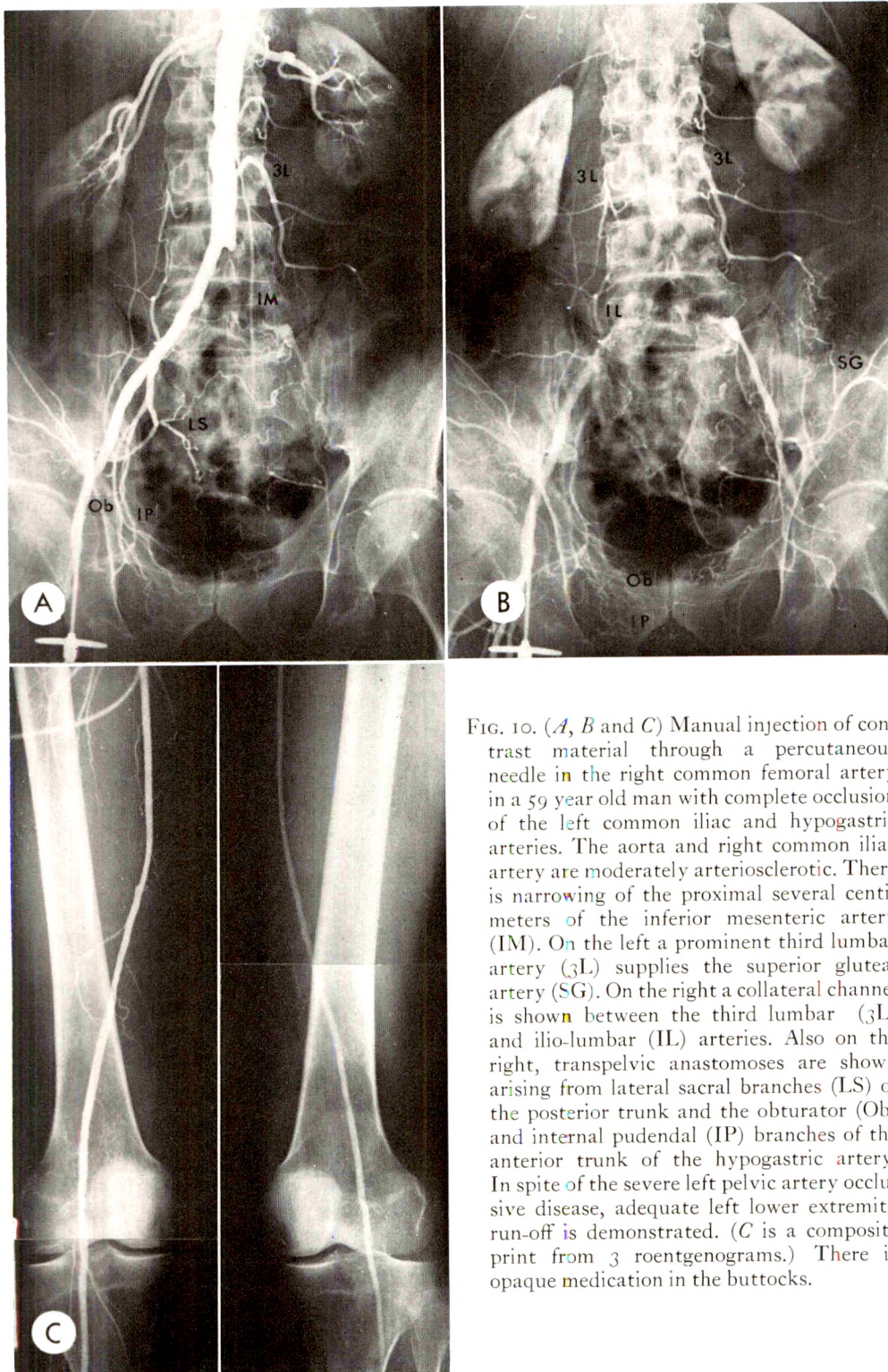


FIG. 10. (A, B and C) Manual injection of contrast material through a percutaneous needle in the right common femoral artery in a 59 year old man with complete occlusion of the left common iliac and hypogastric arteries. The aorta and right common iliac artery are moderately arteriosclerotic. There is narrowing of the proximal several centimeters of the inferior mesenteric artery (IM). On the left a prominent third lumbar artery (3L) supplies the superior gluteal artery (SG). On the right a collateral channel is shown between the third lumbar (3L) and ilio-lumbar (IL) arteries. Also on the right, transpelvic anastomoses are shown arising from lateral sacral branches (LS) of the posterior trunk and the obturator (Ob) and internal pudendal (IP) branches of the anterior trunk of the hypogastric artery. In spite of the severe left pelvic artery occlusive disease, adequate left lower extremity run-off is demonstrated. (C is a composite print from 3 roentgenograms.) There is opaque medication in the buttocks.

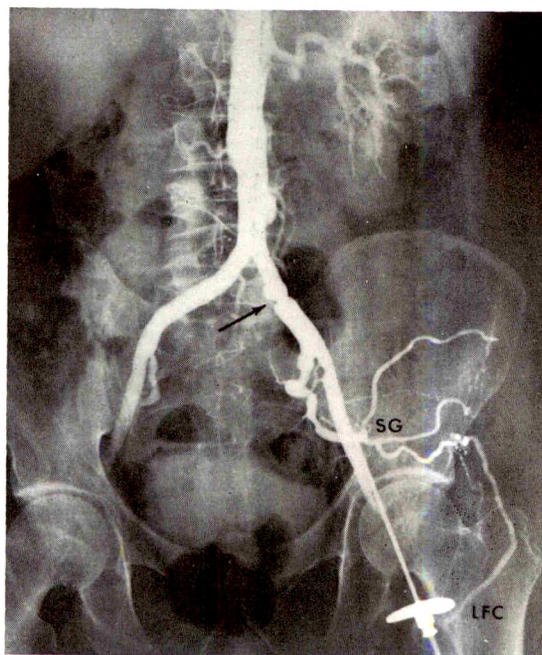


FIG. 11. Manual injection of contrast material through a percutaneous needle in the left common femoral artery in a 63 year old man with a partial segmental occlusion in the mid left common iliac artery (arrow). There are arteriosclerotic plaques in the proximal left external iliac and both hypogastric arteries. The left common femoral artery is completely occluded. There is a well-developed collateral channel between the superior gluteal artery (SG) and ascending branch of the lateral femoral circumflex artery (LFC) to help supply the profunda femoris artery.

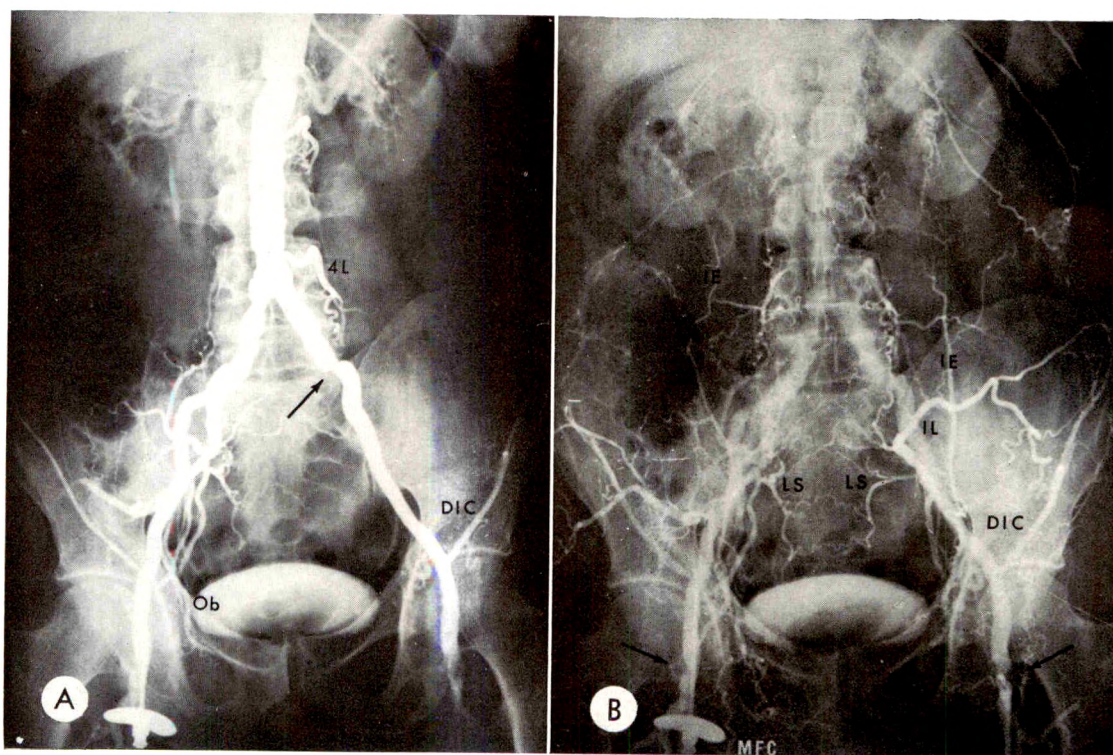


FIG. 12. (A and B) Manual injection of contrast material through a percutaneous needle in the right common femoral artery in a 58 year old man with marked aorto-common iliac arteriosclerosis. There is partial segmental occlusion of the distal left common iliac artery with complete occlusion of the left hypogastric artery (arrow in A). The branches of the left hypogastric artery are supplied by collateral circulation from the opposite lateral sacral artery (LS), as well as collaterals to the ilio-lumbar branch (IL) from the homolateral fourth lumbar (4L) and deep iliac circumflex (DIC) arteries. The inferior epigastric arteries (IE)

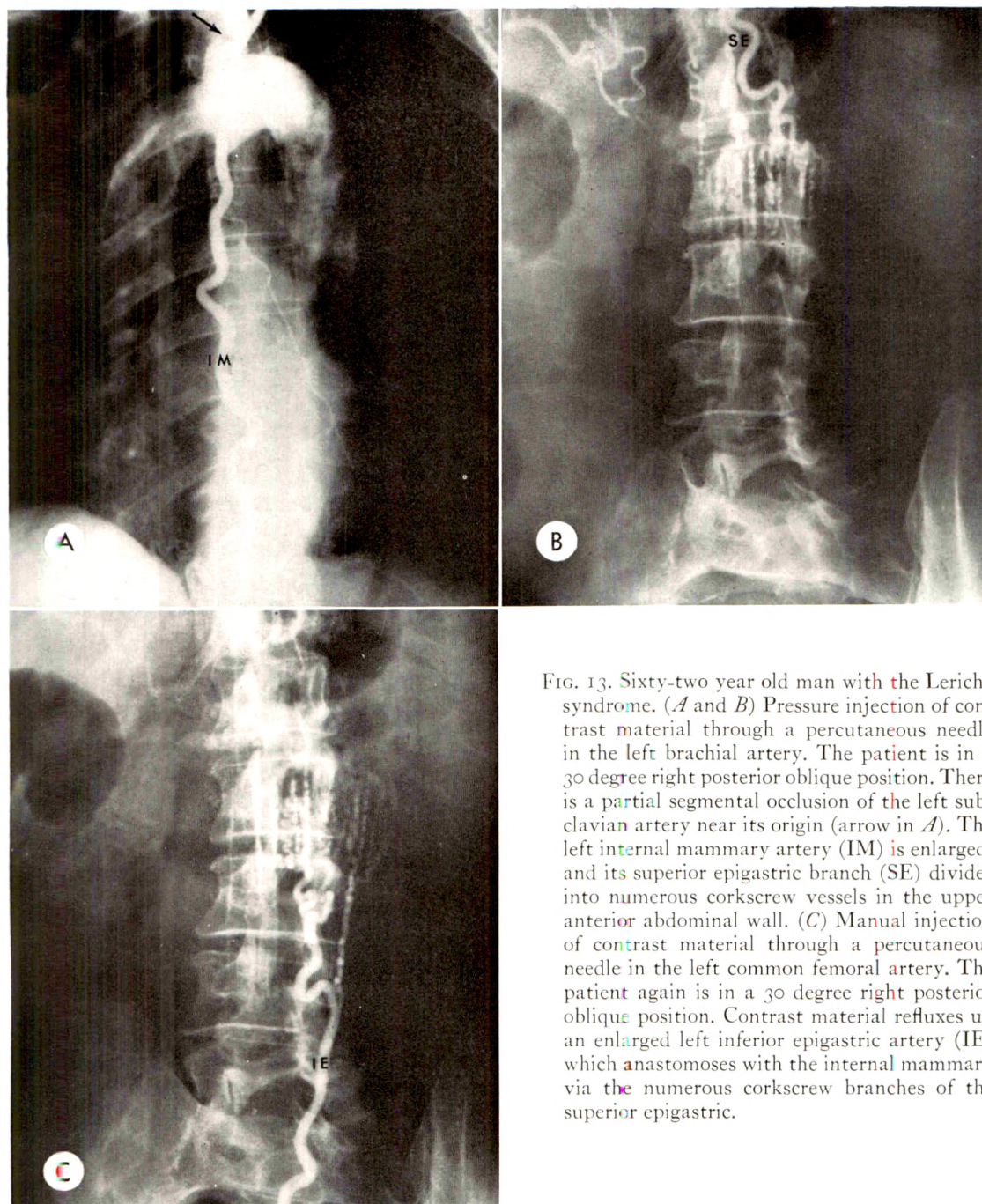


FIG. 13. Sixty-two year old man with the Leriche syndrome. (A and B) Pressure injection of contrast material through a percutaneous needle in the left brachial artery. The patient is in a 30 degree right posterior oblique position. There is a partial segmental occlusion of the left subclavian artery near its origin (arrow in A). The left internal mammary artery (IM) is enlarged, and its superior epigastric branch (SE) divides into numerous corkscrew vessels in the upper anterior abdominal wall. (C) Manual injection of contrast material through a percutaneous needle in the left common femoral artery. The patient again is in a 30 degree right posterior oblique position. Contrast material refluxes up an enlarged left inferior epigastric artery (IE) which anastomoses with the internal mammary via the numerous corkscrew branches of the superior epigastric.

are opacified bilaterally. The left superior gluteal artery is not opacified, probably because of a complete occlusion. There are partial segmental occlusions of the common femoral arteries (arrows in B). On the right there is a collateral channel between the obturator (Ob) and medial femoral circumflex (MFC) arteries.

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COLLATERAL VASCULAR PATHWAYS DURING EXPERIMENTAL OBSTRUCTION OF AORTA AND INFERIOR VENA CAVA*

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FAMILIARITY with the collateral channels which appear when a major blood vessel is experimentally occluded is important for at least two reasons: (a) it clarifies some of the clinical findings observed in obstruction of great vessels in man, and (b) it aids in evaluating the method of experimental major vessel occlusion in which venomotor responses are tested.

The present study represents an attempt to demonstrate the collateral pathways which are utilized under certain special conditions of major vessel occlusion. It also reveals the vasomotor responses of these vessels during stimulation of the carotid sinus and central part of the vagus nerve.

THEORETIC CONSIDERATIONS

Partial obstruction (coarctation) of the aorta causes a difference in systolic blood pressure above and below the stenosis, whereas the diastolic pressures in both areas are in some cases elevated,¹⁸ in others not.¹⁰ The amplitude of pulsation in the abdominal aorta is decreased. The blood flow, although decreased, is frequently sufficient to preserve organ function; in only 25 per cent of the cases reduced organ function is observed.¹⁰ The blood flowing in the aorta beyond the coarctation represents blood which has passed through the stenotic segment and that which flows around the stenotic segment through collateral channels. The contribution of this collateral circulation depends not only on the difference in systolic pressure between the area above and below the coarctation, but also on the total cross-sectional area of all the bridging collaterals. An excellent postmortem study

of the collateral circulation in a cadaver of a patient known to have had coarctation of the aorta has been reported,⁶ as have roentgen studies during experimental obstruction of the aorta in dogs.^{2,12} More information is available concerning the collateral venous circulation following obstruction of the inferior vena cava.^{1,7,9,13-15,17,19,20}

Experimental major vessel occlusion (simultaneous occlusion of thoracic aorta and inferior vena cava) has been utilized to study the changes in inferior vena caval pressure during stimulation of the carotid sinus or of the central end of the vagus nerve.³ Under these circumstances the pressure in the inferior vena cava is increased during reflex stimulation, and this has been attributed to an increase in the muscular tone in its wall (venomotor tone). An alternate explanation for this pressure increase exists if, during the stimulation, it can be shown that there is an increase in flow through the collaterals.

METHODS

Due to the similarity of its topographic vascular anatomy and vascular anastomoses to man,^{8,16} the dog was chosen as the experimental animal. Ten mongrel dogs weighing between 10 and 16 kg. were anesthetized with 30 mg./kg. of pentobarbital intravenously. Blood clotting was prevented by mepesulfate 20 mg./kg. intravenously. The trachea was intubated and respiration was maintained artificially with a pump. Both carotid arteries and vagi were exposed, and the vagi cut. The central end of the right vagus nerve was placed on a pair of electrodes for central vagal

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stimulation. The stimulation parameters (usually 35 v., 20 impulses per sec., and 2 msec. duration) were selected to cause a rise of blood pressure. The chest was opened by midline incision and the internal mammary arteries and veins and the azygos vein were ligated. Two clamps were placed around the aorta, ready for occlusion. One was placed just distal to the left subclavian artery (high occlusion) and the other directly above the diaphragm, usually between the origins of the 8th and 9th intercostal arteries (low occlusion). For venous occlusion an umbilical tape was placed around the inferior vena cava between the diaphragm and right atrium, and another around this vein directly above the entry of the renal veins. Catheters were placed in the aortic arch by way of the left carotid artery and brachiocephalic trunk, in the abdominal or thoracic aorta *via* the femoral artery, and in the inferior vena cava *via* the femoral vein. Diatrizoate sodium (hypaque) 50 per cent was continuously infused into the areas studied at a rate of .64 ml./sec. with an infusion pump. This relatively slow infusion rate was selected to minimize pressure changes incurred from the injection itself. Roentgenography was performed with a Schönander rapid film-changer using variable programming. The factors were 75 to 80 kv., 30 ma., 1/60 second exposure time, and target-film distance of 90 cm.

The ligation of the internal mammary arteries and veins and the azygos vein was performed in order to better demonstrate the other major and minor collateral pathways.

Simultaneous occlusion of the aorta and inferior vena cava was performed for a number of reasons. It had the advantage of demonstrating the collateral leaks into and from the occluded areas. It also slowed the blood flow so that we were enabled to follow the contrast material in the arterial collateral vessels more readily on the serial roentgenograms. It prevented the exaggerated blood pressure increase in the upper circulation which occurs when the descending aorta is occluded with intact venous re-

turn. It prevented the overdilatation of the venous bed which occurs when a major vein is occluded without simultaneous occlusion of the aorta. When both major vessels are occluded, the venous occlusion pressure reaches a level of only 7 to 14 mm. Hg,¹¹ whereas it approaches 80 mm. Hg¹⁴ when the aorta is not occluded simultaneously. The physiologic consequences of major vessel occlusion for short periods of time are discussed elsewhere.³

The following types of experiments were performed:

I. Arterial infusions.

(a) High occlusion of thoracic aorta and simultaneous occlusion of the inferior vena cava.

(1) Infusion just above the aortic occlusion site.

(2) Infusion just below the aortic occlusion site.

(b) Low occlusion of thoracic aorta and simultaneous occlusion of the inferior vena cava.

(1) Infusion just above the aortic occlusion site.

(2) Infusion just below the aortic occlusion site.

II. Venous infusions.

(a) High occlusion of thoracic aorta and simultaneous occlusion of inferior vena cava just above diaphragm; venous infusion below occlusion level.

(b) High occlusion of thoracic aorta and simultaneous occlusion of inferior vena cava just above diaphragm and just above renal veins.

(1) Infusion below lowest venous occlusion level.

(2) Infusion between venous occlusion sites.

RESULTS

I. ARTERIAL INFUSIONS

Studies of the arterial collaterals were performed in 10 open-chest dogs. Figure 1 shows the aorta and intercostal and lumbar arteries. The original roentgenogram shows

the side branches of the intercostal arteries forming anastomotic bridges between them and also the junction of the anterior and posterior intercostals at the end of each rib. The posterior intercostals are supplied by the aorta, the anterior intercostals by the internal mammary arteries.

(a) *Arterial infusion during high occlusion of thoracic aorta and simultaneous occlusion of the inferior vena cava.*

The diagram in Figure 2 demonstrates the routes taken by the contrast medium to bypass the aortic occlusion as established by serial roentgenograms. The aortic blood above the occlusion enters both the posterior intercostal arteries and spinal arteries. In this experiment the 3rd and 4th posterior intercostal arteries were filled via the 5th posterior intercostal as shown in Figure 1. From the posterior intercostals, the blood flows in reverse direction through the anterior intercostals into the internal mammary. From here the blood flows into the posterior intercostals below the clamp and then into the aorta. Blood is also supplied to the intercostals below the clamp from other anastomoses originating in the posterior intercostals, subscapular and lateral thoracic vessels above the clamp.

In 3 other experiments with the same type of occlusion but with the contrast medium injected in the left subclavian artery, the reverse flow in the posterior intercostals below the clamp was maintained by blood originating: (1) from the supreme intercostal arteries, (2) from the many anastomoses existing between the upper posterior intercostals (see right side of Fig. 1), and (3) from the anterior intercostals as part of the internal mammary system. Another collateral pathway is that of the spinal arteries. In all experiments the celiac and renal arteries became visible after 6 seconds, filling with contrast medium from the aorta; this fact indicates that contrast medium has accumulated in the aorta below the occlusion site.

To confirm the reversal of flow toward the aorta in the intercostal arteries below

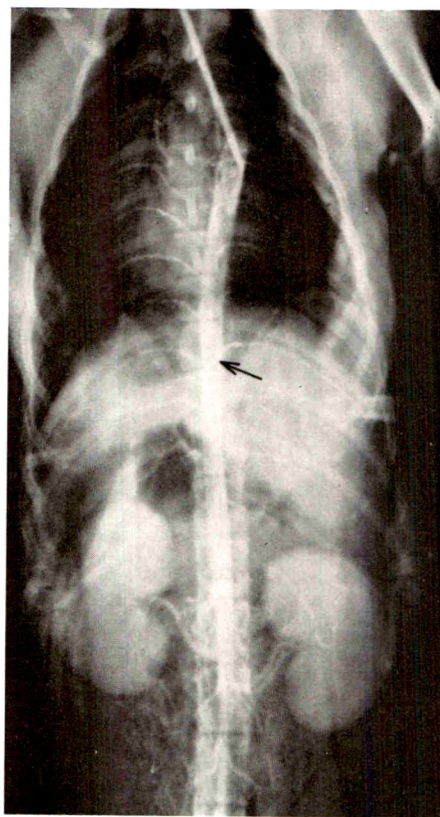


FIG. 1. Arteriogram of the unoccluded aorta of the dog after 2 seconds of infusion into the proximal descending thoracic aorta. Note the aortic diameter under normal pressure and the flow of contrast medium on the right to the 2nd, 3rd, and 4th posterior intercostal arteries via the 5th intercostal. The arrow indicates the position of the "low" clamp in Figures 5 and 6. Left side of figure is right side of dog.

the clamp during high occlusion, 4 experiments were performed in which contrast medium was infused into the aorta just below the clamp. Figure 3 shows that the diameter of the aorta is smaller (compare with Fig. 1). The higher intercostals (4th to 8th) do not fill with contrast medium because the flow of blood is toward the aorta. The renal arteries are visualized first and then, after a delay of 2 seconds, the celiac trunk. The superior mesenteric artery and the three lowest intercostals, which fill from the aorta, appear next. Finally, the lower lumbar arteries are seen. In one experiment high occlusion of the aorta was made without occlusion of the

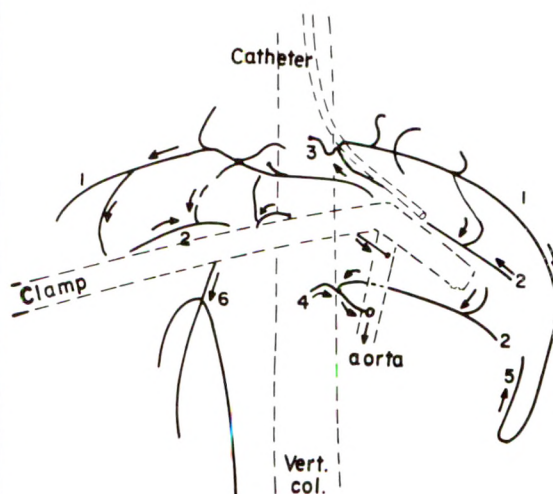
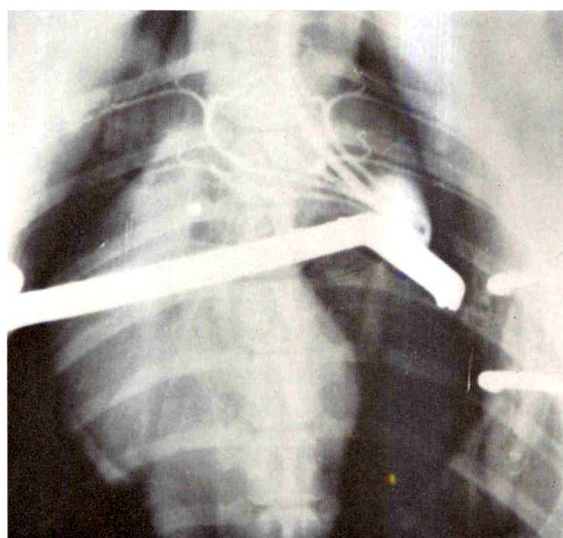


FIG. 2. "High" occlusion of aorta. Roentgenogram was taken after 4 seconds of constant infusion of contrast medium into aorta above the clamp. The diagram shows the spread of contrast medium from the area above the clamp to the area below the clamp. The arrows indicate the direction of blood flow established by serial roentgenograms. (1) Posterior intercostal above clamp; (2) posterior intercostal below clamp; (3) spinal branch above clamp; (4) spinal branch below clamp; (5) anterior intercostal (from internal mammary); (6) pericardial branch (from internal mammary).

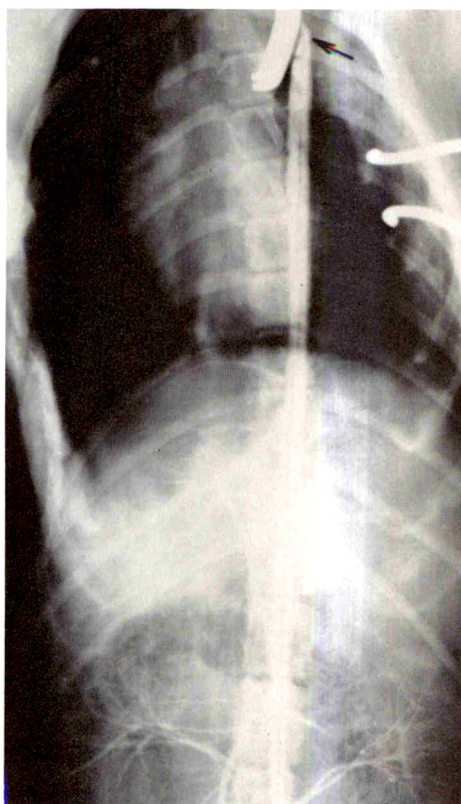


FIG. 3. "High" occlusion of aorta. Roentgenogram was taken after 10 seconds of constant infusion of

inferior vena cava. The venous drainage therefore was quicker and we assume the arterial passage was more rapid because of the greater arteriovenous pressure gradient. Under this condition the lower lumbar and the three lowest intercostal arteries visualized first, then the renal arteries, and, finally, the celiac and mesenteric arteries.

The velocity of the blood flow in the occluded descending aorta can be estimated by the rate of disappearance of contrast medium from this area after the infusion is discontinued. Within 1 to 2 seconds the descending aorta is clear from the site of injection (close to the aortic arch) to the diaphragm.

Figure 4 is a diagram of the arterial collaterals during high obstruction of the aorta (clamp just distal to the left subclavian artery), indicating the preferred direction of flow.

contrast medium into the occluded aorta just below the clamp (arrow points to catheter tip). Note the absence of contrast medium in the posterior intercostal arteries 4 to 8 and the opacification of intercostals 9 through 12.

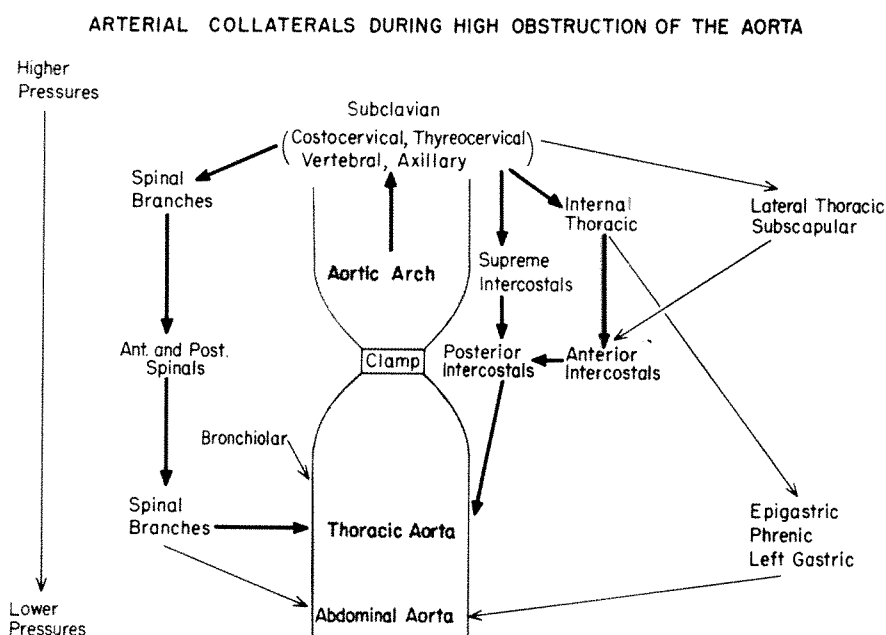


FIG. 4. Diagrammatic representation of the arterial collaterals when the aorta is occluded just distal to the left subclavian artery (high occlusion). Heavy lines indicate the direction of preferred flow.

(b) *Arterial infusion during low occlusion of the thoracic aorta and simultaneous occlusion of the inferior vena cava.*

During low occlusion, in 3 of the 4 experiments the contrast medium infused into the aorta above the clamp entered all the aortic intercostal arteries above the clamp (Fig. 5). In the fourth experiment it entered only intercostals 7 and 8. Figure 5 demonstrates the various collateral routes. The arrival of contrast medium in the aorta below the clamp is signified by the faint outline of the celiac trunk and the right renal artery. In all experiments with low occlusion, the contrast medium arriving below the clamp first appeared in the spinal branches of the lowest intercostal arteries, spreading toward the aorta and toward the periphery in the intercostal arteries. The lateral thoracic artery fills with contrast medium from vessels near the ventral tips of the 4th to 8th ribs as indicated in Figure 5. We were able to recognize this vessel in all experiments with low occlusion but not in the ones with high occlusion. The superior epigastric artery, a branch of the internal mammary, also car-

ries contrast medium caudad.

Figure 6 shows the arterial outlines during low occlusion (contrast medium infused below the clamp). The important findings are that the lateral thoracic arteries and their connections below the clamp did not visualize in any of the 3 experiments (see Fig. 5), and that the lumbar arteries are not visualized because both arterial systems are conducting blood toward the aorta below the clamp. The larger vessels originating from the abdominal aorta, namely, the renal, mesenteric, celiac, and lowest three intercostal arteries, appear as they do in the normal animal.

Figure 7 is a diagram of the various collateral pathways during low obstruction of the aorta.

The relative rate of the collateral flow with low occlusion can be estimated from the time the medium first appears in the renal artery. In the four experiments it took 12, 22, 25, and 35 seconds, respectively, compared with the much shorter times in the high occlusion experiments where we found times of 5, 6, 10, and 11 seconds, respectively (the same 4 dogs

were used for both high and low occlusions). Another indication of the slower flow during low occlusion is the much slower clearance time of the contrast medium in the aorta below the clamp after the infusion had been discontinued. It took about 15 seconds to clear the aorta for a length of one intercostal space, compared with 1 to 2 seconds with high occlusion.

II. VENOUS INFUSIONS

In 8 experiments with high thoracic aorta occlusion, contrast medium was infused into the inferior vena cava. As stated, in all experiments the azygos vein and internal mammary veins were ligated beforehand. The constant infusion of contrast medium at a rate of .64 ml. per second increased the venous pressure by only .4 to

1.4 mm Hg per minute. Such a small increase of the venous occlusion pressure during infusion cannot be the driving force which controls the direction of flow of the contrast medium.

(a) *High occlusion of thoracic aorta and simultaneous occlusion of inferior vena cava just above diaphragm; venous infusion below occlusion level.*

A summary of the venous collaterals between the inferior and superior vena cava is shown in Figure 8. This diagram is based on observations made during serial filming. Contrast medium appears in the following order during continuous infusion: 5 seconds after start of infusion the pair of lower lumbar veins, the precentral vein, the vertebral venous plexus up to the 10th intercostal space, and the higher pairs of

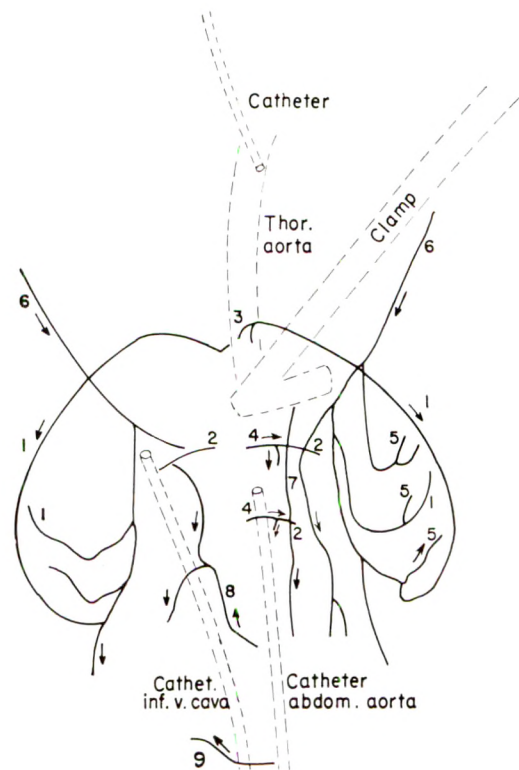
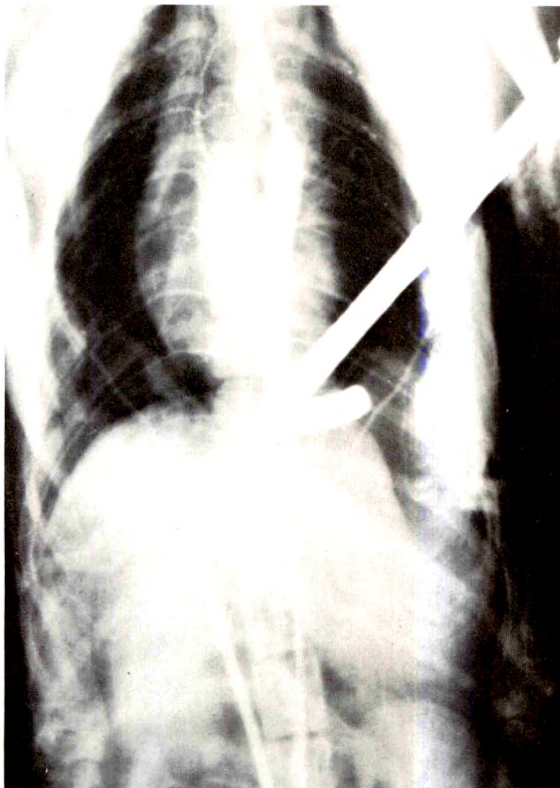


FIG. 5. "Low" occlusion of the aorta. Roentgenogram was taken after 8 seconds of constant infusion of contrast medium into the aorta above the clamp. The diagram shows the flow of contrast medium with arrows indicating the direction as established by serial roentgenograms. (1) Posterior intercostal above clamp; (2) posterior intercostal below clamp; (3) spinal branch above clamp; (4) spinal branch below clamp; (5) anterior intercostal (from internal mammary); (6) lateral thoracic artery; (7) superior epigastric (from internal mammary); (8) celiac trunk; (9) renal artery.

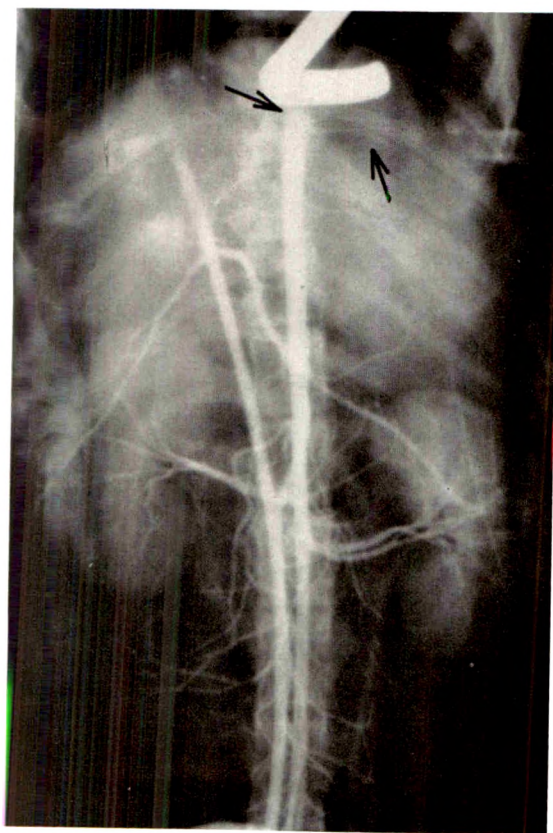


FIG. 6. "Low" occlusion of aorta. Roentgenogram was taken after 10 seconds of constant infusion of contrast medium into the occluded abdominal aorta just below the clamp (left arrow points to catheter tip). Another catheter locates the position of the inferior vena cava. Right arrow: 10th intercostal artery. Note the absence of contrast medium in the branches of the internal mammary artery and the spinal branches of the lumbar arteries.

lumbar veins are outlined; after 10 seconds, the occluded azygos vein, intercostal veins 10 to 12, the subcostal veins, and the

vertebral venous plexus up to the 5th intercostal space; and after 15 seconds, the highest portion of the vertebral venous plexus, the higher intercostal veins including their chest wall anastomoses with the lateral thoracic, subscapular and superior and supreme intercostal veins.

(b) *High occlusion of thoracic aorta and simultaneous occlusion of inferior vena cava just above diaphragm and just above renal veins.*

(1) Infusion below lowest venous occlusion level. In order to better evaluate the

ARTERIAL COLLATERALS DURING LOW OBSTRUCTION OF THE AORTA

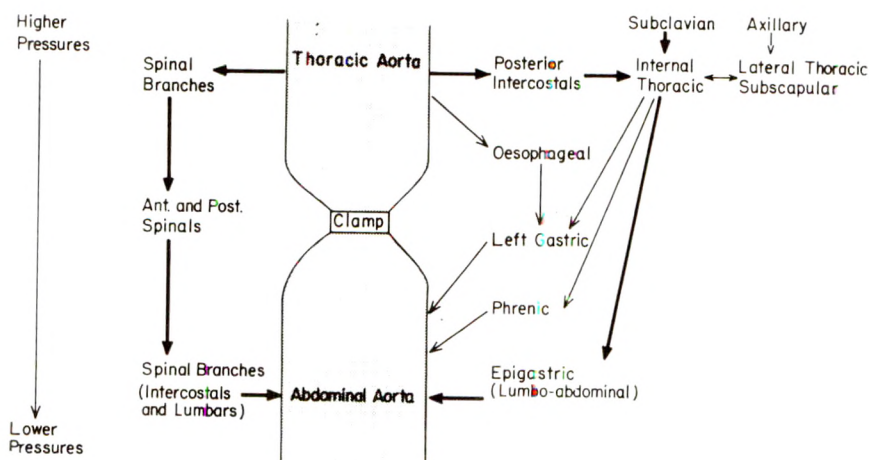


FIG. 7. Diagrammatic representation of the arterial collaterals when the aorta is occluded between the origin of the 8th and the 9th posterior intercostal arteries (low occlusion). Heavy lines indicate the direction of preferred flow.

collateral pathways, this segmental occlusion of the inferior vena cava was performed. Extension of the contrast medium into the region of the hepatic veins was prevented by the ligature directly above the renal veins (Fig. 9). The lumbar veins shunt contrast material from the inferior vena cava to the vertebral venous plexus. Above the lowest occlusion site, the two next higher pairs of lumbar veins shunt blood from the vertebral venous plexus to the azygos vein and from here the lower intercostal veins fill. The origin of the azygos vein appears as a straight vessel, the precentral or ascending lumbar vein, which extends upward from the region of the lower lumbar veins.

The flow of the contrast medium in the same experiment was followed by serial roentgenography. Figure 10 demonstrates the upper part of the venous circulation at a later time of continuous infusion. The occluded azygos vein is shown with intercostal veins filling from it. The vertebral venous plexuses continue to carry contrast medium. The lateral thoracic vein and subscapular vein fill via anastomoses with the lower intercostal veins. The lateral thoracic and subscapular veins drain into the axillary veins. The higher intercostal veins drain to the right superior intercostal vein,

the left draining directly into the brachiocephalic vein is not visible in Figure 10.

Thus, the 5 major shunts between the inferior and superior vena cava are: (a) the azygos vein; (b) the internal mammary veins (both of these systems were occluded in this experiment); (c) the continuation of the blood up the vertebral venous plexus with final drainage through the vertebral, costocervical and occipital veins to the superior vena cava; (d) the higher intercostal veins to the superior intercostal veins, the left draining into the subclavian vein, the right, since the azygos is occluded, draining into the right supreme intercostal vein; and (e) the lower intercostal veins through the chest wall to the lateral thoracic and subscapular veins draining into the axillary veins.

(2) Infusion between the venous occlusion sites. In the case shown in Figure 11, the contrast medium was infused into the hepatic segment of the inferior vena cava and there is no indication of anastomoses from this portion of the vessel. Our infusions were not continued long enough to show the anastomoses connecting the portal circulation with adjacent venous areas.¹⁷

In all experiments with infusion of contrast medium into the inferior vena cava,

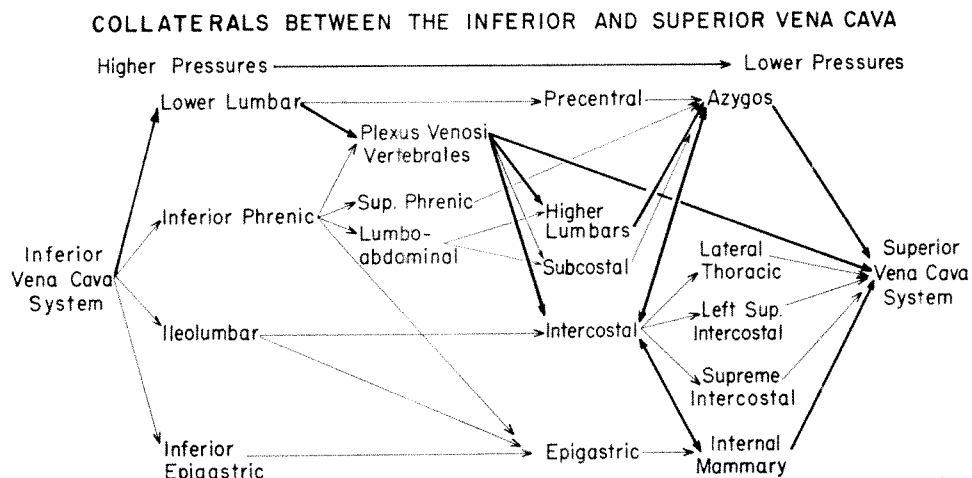


FIG. 8. Diagrammatic representation of the venous collaterals when the inferior vena cava is occluded just above the diaphragm. Heavy lines indicate the direction of preferred flow.

information was obtained concerning the relative speed of blood flow in this area. In one experiment medium was infused for only 3 seconds into the inferior vena cava. The lumbar veins and vertebral venous plexus contained contrast medium after 5 seconds and these vessels were clear again after 10 seconds. On the other hand, the clearance of medium from the inferior vena cava, after infusions of longer duration were discontinued, took place in 5 to 21 seconds. The central end of the vagus nerve was stimulated in order to increase blood pressure in all areas. The diameter of the venous vessels was not influenced by

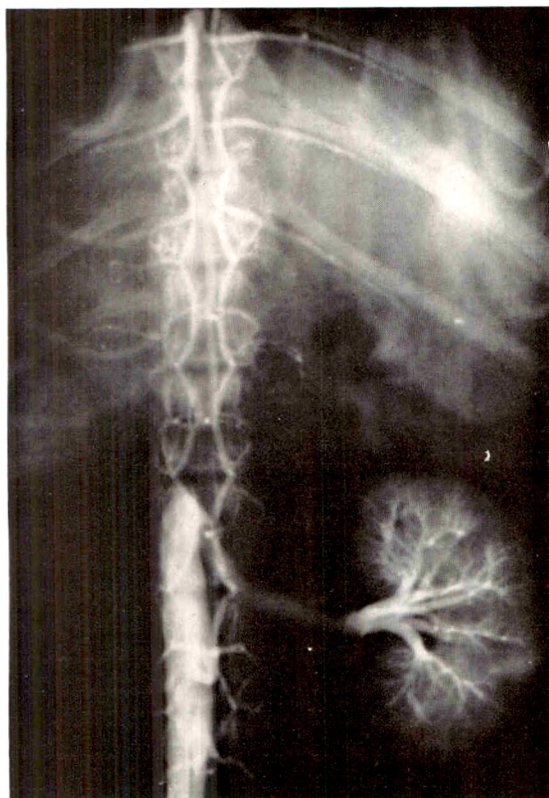


FIG. 9. Contrast medium infused into the lower inferior vena cava, occluded in the thoracic cavity and just above the renal veins. Roentgenogram was taken 15 seconds after start of infusion. Outlined are lower lumbar veins connected with inferior vena cava, higher lumbar veins connected with azygos vein (top of figure over right side of vertebral column); vertebral venous plexus (garland-like double row of vessels over vertebral column) and lower intercostal veins.

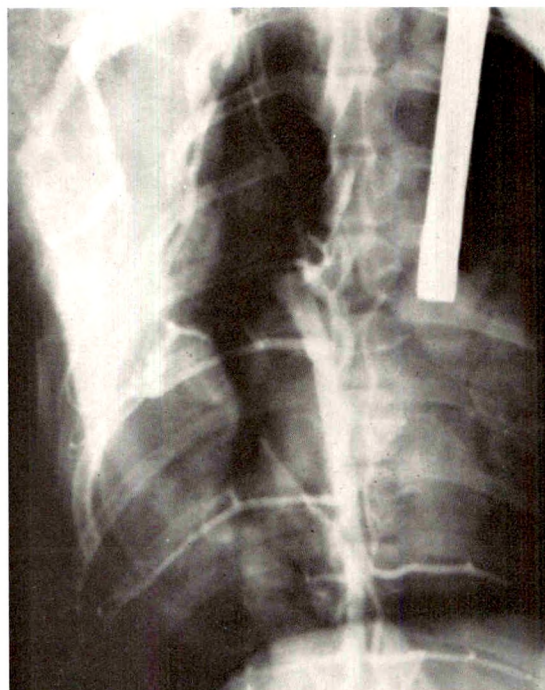


FIG. 10. Same experiment as in Figure 9. Roentgenogram was taken after 35 seconds of contrast medium infusion. Outlined are occluded azygos vein, right superior intercostal vein, intercostal veins with anastomoses to lateral thoracic and subscapular vein and vertebral venous plexus.

this procedure but the inferior vena cava was cleared faster of contrast medium; the clearance time was 3 to 10 seconds.

DISCUSSION

The internal mammary arteries are recognized as the major collateral arterial channels in cases of obstruction of the thoracic aorta.^{2-6, 8, 12, 16, 21} The flow charts in Figures 4 and 7 show the ramifications of this artery. In our experiments we deliberately occluded the internal mammarys in order to study the other bypasses. This prevented blood flow in the natural direction, but did not prevent the collateral blood from utilizing the internal mammary bed (Fig. 5).

The study supplied evidence of 2 other major bypasses in the dog (Fig. 4): (1) the extensive collaterals between the supreme intercostal arteries, which form the first 2 and sometimes 3 posterior intercostal

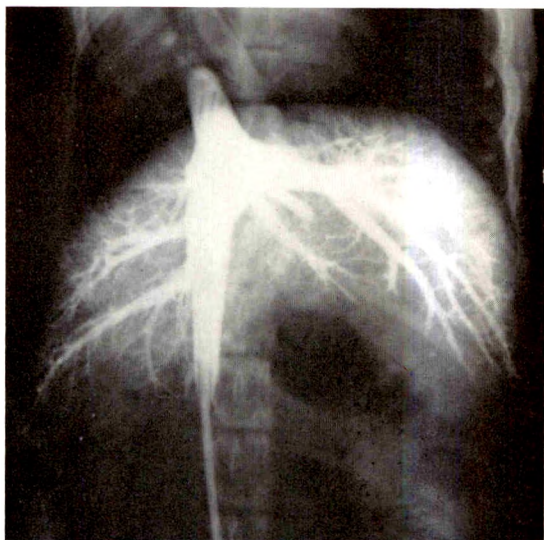


FIG. 11. Contrast medium infused into inferior vena cava between clamp placed in the thoracic cavity and just above the renal veins. Roentgenogram was taken after 55 seconds of infusion. Only the hepatic veins fill with contrast medium.

arteries, and the 4th intercostals, which originate as the first pair from the aorta and feed the 2nd and 3rd intercostals. Numerous anastomoses exist between all the intercostals via sternal and spinal branches. Only the last 3 posterior intercostals (10 to 12) are less favored by collaterals; and (2) the anterior and posterior spinal arteries are amply connected with all intercostal, lumbar, and sacral arteries.

In accordance with this distribution of anastomoses, an occlusion of the aorta close to the origin of the left subclavian artery was accompanied by a larger amount of collateral blood flow (faster clearance), while smaller collateral flow (slower clearance) occurred when the aorta was occluded in the vicinity of the diaphragm. In the latter case the collateral blood flow was mainly *via* the spinal arteries, the lower branches of the internal mammary (musculo-phrenic and epigastric arteries), and the esophageal arteries. Accordingly, the 10th and 12th intercostal arteries and subcostal arteries did not show reversed flow. With low occlusion the lateral thoracic arteries were filled with contrast

medium from above the occlusion, but we could not detect any direct anastomoses between these vessels and the lower intercostal arteries, although the shunts provided by the internal mammary bed were used (see Fig. 5).

These differences in collateral blood flow between high and low obstruction of the aorta are substantiated by the time of appearance and disappearance of contrast medium in the respective areas. Indirect evidence is available from pressure measurements and from the collection of plasma tagged with dye.¹¹

In short, the largest collateral flow exists when the aortic obstruction is in the region of the aortic arch. *The lower the obstruction, the less the collateral blood flow.* From blood collection through large catheters placed into the abdominal aorta *via* the femoral arteries during obstruction of the thoracic aorta at different sites, we estimate the collateral blood flow during the highest occlusion (with simultaneous occlusion of the inferior vena cava) to be around 10 per cent of the normal flow and with the low occlusion to be around 3 per cent.¹¹ Allbaugh and Horvath² observed collateral blood flow of 35 to 40 per cent during obstruction, but in their experiments the venous return was unobstructed and the internal mammary arteries and veins were open.

Our studies give some insight into the reasons why the highest and lowest intercostal arteries usually do not cause rib notching in aortic coarctation.⁵ The upper intercostals (1 to 3) rarely carry collateral backflow because they originate above the obstruction in the high pressure area. In addition, the shunt between supreme intercostals and aortic intercostals is located close to the vertebral column, the intercostal arteries 1 to 3 being merely side branches of this major anastomosis in both dog and man. Enlargement of the arteries is not caused by high pressure but by high blood flow. The lowest posterior intercostals (10 to 12) are not distended enough to cause rib notching. Again there is no backflow in these arteries because there

are no anterior intercostals for anastomosis. They, therefore, do not receive collateral flow from areas above the aortic obstruction. The only parts of these vessels that carry collateral blood flow are the short spinal branches between the spinal arteries and the origin of the posterior intercostal arteries from the aorta.

The venous collaterals during major venous obstruction have to be considered in two different areas. In an isolated high obstruction of the inferior vena cava, the venous blood has 3 major avenues: (1) the vertebral venous plexuses; (2) the azygos system; and (3) the internal mammary veins. The third avenue is not demonstrated in our experiments because these vessels were ligated. Contrast medium infused into the occluded inferior vena cava reaches the vertebral venous plexuses either through the lower lumbar veins or through the phrenic veins to the dorsal branches of the plexuses (Fig. 8). An excellent anatomic study of these pathways is available.⁷ From here the contrast medium can enter the azygos system *via* the upper lumbar veins or *via* the dorsal branches of the posterior intercostal veins. In addition, the inferior vena cava communicates with the azygos through the ilio-lumbar veins and the 2 lowest intercostal veins, through the phrenic and lumbo-abdominal vein to the subcostal vein, or directly through the precentral or ascending lumbar vein. Assuming the appearance and clearance of contrast medium in the different collateral bypasses is indicative of the preferred direction of flow, the quickest exchange takes place in the lumbar veins, the precentral vein, and the vertebral venous plexuses. The other pathways clear more slowly.

Having reached the venous circulation above the diaphragm, the venous drainage favors the azygos vein, the internal mammary vein, and the venous plexuses. Since we occluded the azygos and internal mammary veins, minor shunts became apparent: the venous blood drained through the venous plexuses or through

the superior intercostal veins, the right communicating with the supreme intercostal vein and the left draining directly into the subclavian vein; in addition, the venous blood flowing in reverse direction through the intercostal veins is drained by the lateral thoracic and subscapular veins, ending in the axillary veins.

Keen¹³ suggested that parts of the collateral venous vessels which develop during chronic obstruction of the inferior vena cava are distentions of primitive, embryonic venous pathways like the left posterior cardinal vein. A major argument for this view is the observation that the right side of the venous circulation is more embarrassed (*i.e.*, edema, greater venous pressure, etc.) than the left following caval obstruction.¹⁷ This is because the inferior vena cava represents a composite of the embryonic vessels on the right and consequently, when it is obstructed, only the left primitive veins can reopen to facilitate drainage.

We propose an alternative explanation for this phenomenon, namely, that the natural venous collaterals are sufficient to account for the enlarged new venous pathways during inferior vena cava obstruction. On caval ligation the venous blood below the obstruction reaches the azygos vein (right side) through the major shunts outlined in Figure 8. This increased blood flow in the azygos vein creates increased resistance to the drainage of the secondary collaterals of the right side, which terminate in the intercostal veins and the azygos vein as well. This overloading of the azygos vein leads to enlargement of collaterals leading to the axillary and subclavian veins. Unfortunately, the right superior intercostal vein drains into the azygos too, and hence this vein plays little role in collateral blood flow. On the left side, on the other hand, the secondary collaterals can drain freely into the hemiazygos and accessory hemiazygos veins. The collaterals to the axillary and subclavian vein are not overloaded. In addition, the left superior intercostal vein draining directly into the left

brachiocephalic vein provides an additional channel for easy collateral drainage of the left side. Therefore, there is no need to invoke the reopening of embryonic pathways for the establishment of venous collateral blood flow.

SUMMARY

In order to study arterial and venous collateral pathways in the dog, radiopaque material was continuously infused and was followed by fast roentgenography during simultaneous obstruction of the aorta and the inferior vena cava.

Total obstruction of the descending aorta and the inferior vena cava, accompanied by occlusion of the azygos vein and the internal mammary arteries and veins does not prevent the continuation of blood flow through the obstructed areas, although it does greatly reduce this flow.

The arterial collateral flow becomes smaller the greater the distance of the site of the aortic occlusion beyond the origin of the left subclavian artery. The major collateral pathways around high and low aortic occlusions are summarized in Figures 4 and 7. The reason for the lack of enlargement of the two highest and three lowest posterior intercostal arteries (rare occurrence of rib notching) is that these vessels carry less collateral blood flow.

Venous blood flow from the occluded inferior vena cava and azygos systems continues to reach the superior vena cava by the pathways summarized in Figure 8. The anatomic site and the availability of the venous collaterals suggest that these existing vessels may be enlarged when the inferior vena cava, the azygos vein, or the superior vena cava is chronically obstructed. There is no need to invoke the reopening of lost embryonic pathways for the establishment of venous collateral blood flow. During inferior vena cava obstruction, the left side is less embarrassed than the right because of the additional channel on the left, namely, the left superior inter-

costal vein draining directly into the brachiocephalic vein.

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INTRA-OSSEOUS COSTAL VENOGRAPHY*

ASSESSMENT OF THE METHOD IN STUDIES ON PORTAL HYPERTENSION

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IN THE investigation of patients with presumed portal hypertension, percutaneous splenoportography is of central importance and usually gives valuable information regarding the pathologic and hemodynamic conditions. In certain cases, however, *e.g.*, following splenectomy, the procedure is inapplicable. Moreover, it is not suitable for frequent follow-up studies.

Following the suggestion of Ehrhardt and Kneip² to inject contrast medium directly into the medullary cavity of bones, a number of methods dealing with the application of this procedure in studying various venous pathways were published. In 1954 Tori¹⁴ and Süsse and Aurig¹³ suggested the injection of contrast medium into a rib in order to visualize the azygos system. Since 1957, Schobinger and co-workers^{5,10} have shown how hemodynamic alterations under various morbid conditions, especially portal hypertension, might be reflected in individual azygographic patterns characteristic of certain disease entities. These venous patterns have been illustrated by Schobinger in numerous examples, but the papers do not contain an exact analysis substantiating the claimed value of the method.

At the Copenhagen County Hospital in Gentofte, Denmark, the procedure was assessed with a particular view to its applicability in evaluating portal hypertension.

METHOD

The technique corresponds accurately to that advocated by Schobinger. With the patient supine and under local anesthesia, a bone-marrow needle is introduced into the 10th, rarely the 9th, rib in the left midaxillary line, and 20 ml. of contrast medium

(urografin 60 per cent) is injected intracostally in 3 to 5 seconds. A single roentgenogram made shortly before the injection is completed may be sufficient, but in order to be able to fully assess the value of the method, we made 4 to 8 exposures at $\frac{1}{2}$ second intervals at the termination of the injection. In order to avoid a Valsalva effect, the patient is instructed to relax, breathe quietly through his mouth, and to stop breathing without straining immediately before the exposures. The injection may elicit a very short-lasting sensation of pain, but otherwise there are usually no complaints during or after the procedure, which is technically very easy.

In the first one-third of our patients, we injected procaine intracostally immediately before the contrast medium was introduced, as recommended by Schobinger, but, since the value of this injection is problematic, it was omitted in the latter two-thirds.

A small amount of contrast medium may run back along the needle and be deposited paracostally. This may give rise to tenderness at the site of puncture. No other complications were observed.

ANATOMY OF THE AZYGOS SYSTEM

Between the superior and inferior venae cavae, there are several communications, the largest and most direct being the azygos system.

The azygos vein arises inferiorly as the continuation of the right ascending lumbar vein which again communicates with the right common iliac vein. At the level of the 4th-5th dorsal vertebrae, it curves anteriorly above the right main bronchus and opens

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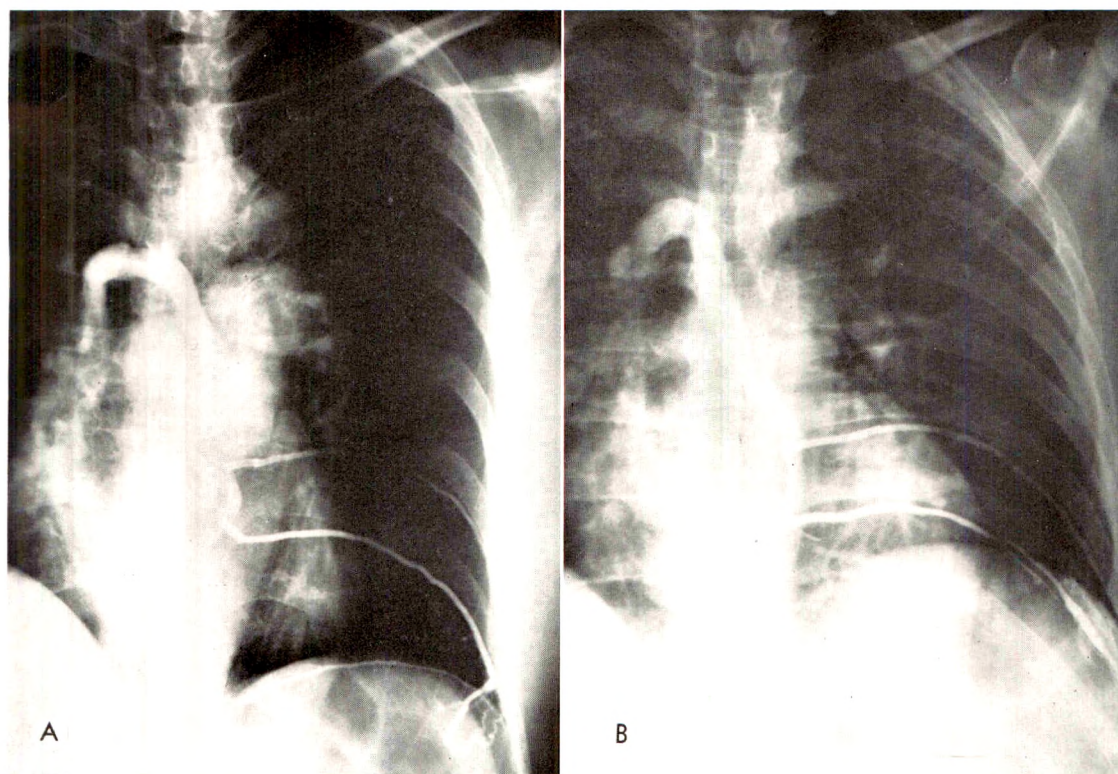


FIG. 1. Normal azygograms. (A) Sixty-two year old female without clinical suspicion of portal hypertension or heart disease. (B) Forty-six year old male with confirmed portal hypertension.

into the superior vena cava. It receives blood from all the intercostal veins on the right side. On the left of the spine, the hemiazygos vein inferiorly communicates with the ascending lumbar vein, the common iliac vein, and often also with the renal and spermatic veins. The hemiazygos vein enters the azygos vein on a level with the 7th-9th dorsal vertebrae. Its tributaries are the lowest 4 to 6 intercostal veins on the left. The remaining left-sided intercostal veins empty directly or indirectly into the accessory hemiazygos vein, which passes downward on the left of the spine and opens into the azygos or hemiazygos vein. Variations are very common.

Contrast medium injected into the 10th or 9th rib on the left will immediately pass into one or two intercostal veins, corresponding to the level of injection. Hence, it is carried to the hemiazygos vein, and further, by way of the azygos vein, to

the superior vena cava. This simple venous pattern Schobinger calls the normal azygogram, which, according to him, is a regular finding in all normal individuals (Fig. 1, A and B).

In the event of altered hemodynamics, the contrast medium will be carried by other routes, and Schobinger has described 3 venographic patterns which he relates to well-defined disease entities.

Splenoportographic studies of portal hypertension have demonstrated how the blood flow in the portal venous bed may be reversed into a hepatofugal direction, usually flowing toward the esophageal veins. Hence, it usually passes via the azygos vein to the superior vena cava. The greatly increased flow through the azygos vein is stated to be the probable cause of the most important differential diagnostic criterion of portal hypertension in costal azygography, *i.e.*, a very faint or, as a rule, completely lacking opacification of the azygos

vein, although the forward-directed, upper part—the so-called bulb of the azygos vein—is always visible. A simultaneous elevation of the pressure in the azygos system is presumed to explain the presence of one or more of the other characteristics which comprise increased passage of contrast medium through the medullary cavity of the injected rib, visualization of more than 2 intercostal veins and of the lateral thoracic vein, occasionally visualization of veins to the anterior abdominal wall, an increased number and augmented tortuosity of veins at the junction of intercostal and hemiazygos veins, and visualization of the internal vertebral venous plexus (Fig. 2, *A* and *B*). According to Schobinger, the costal azygogram found in patients with portal hypertension is so characteristic that the diagnosis is said to be possible even before esophageal varices are demonstrable on barium swallow studies.

The other two characteristic venographic patterns have been described in obstruction of the azygos vein (by a primary tumor centrally in the right lung or in the middle third of the esophagus or by lymph node metastases in the mediastinum) and cardiac insufficiency with right inflow stasis or constrictive pericarditis, respectively. Both are characterized by the outlining of a varying number of collaterals.

When the obstruction is complete, as in the superior vena cava syndrome, the azygos vein or its bulb will not be visualized at all, but the contrast medium courses—mainly via the ascending lumbar veins—to the inferior vena cava. If there is only partial compression, the azygos vein may be densely opacified to the point of obstruction, usually with delineation of the compressing mass. In cardiac decompensation the reduced flow within the venae

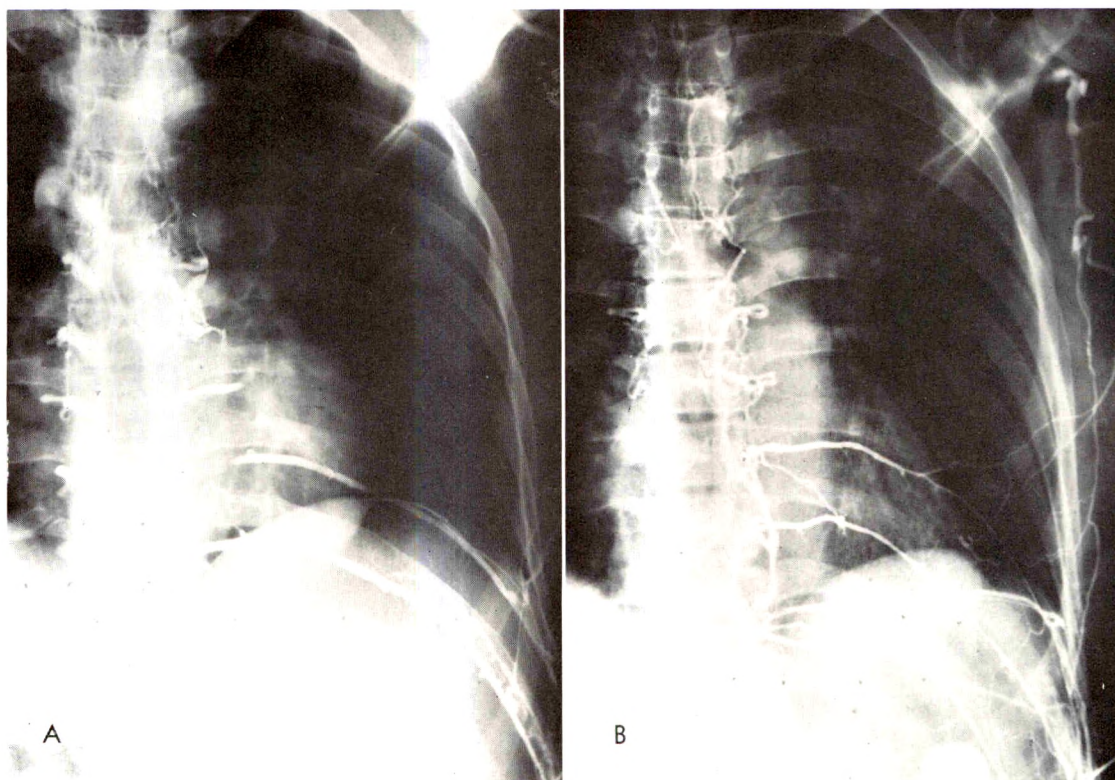


FIG. 2. Abnormal azygograms, increased flow. (*A*) Thirty-eight year old male without clinical suspicion of portal hypertension or heart disease. (*B*) Fifty year old female with confirmed portal hypertension.

TABLE I
AGE DISTRIBUTION AT THE TIME OF
102 COSTAL VENOGRAPHIES

Age	No. of Patients
Under 30	6
30-49	26
50-69	58
Over 69	12

cavae and their related venous territories may cause a very dense opacification of the azygos venous system, a frequent visualization of the superior vena cava, and faint opacification of the right atrium. Similar venograms may be seen when using the Valsalva maneuver.

Thus, the three pathologic venograms are distinguished primarily by the varying opacification of the azygos vein and, secondarily, by the different collaterals.

In portal hypertension complicated by ascites, the azygogram will differ, according to Schobinger,⁸ there being a somewhat better opacification of the azygos vein than observed in portal hypertension without ascites.

Following a successful, surgically established portosystemic shunt, the abnormal azygogram should become normal within a few weeks. Thus, costal azygography is convenient to verify the patency of an artificially created shunt.

MATERIAL

The present material comprises 102 investigations on 80 patients, 43 males and 37 females, whose age distribution is shown in Table I.

We took particular interest in the appearances in intra- or extrahepatic block leading to portal hypertension. Twenty-one patients with confirmed portal hypertension of intrahepatic origin were investigated immediately prior to portosystemic shunt operation. The hypertension was confirmed during the operation, as it was either evident on sight or palpation (8 cases) or demonstrated by direct pressure

measurement (13 cases). Moreover, 13 of the patients had preoperative splenoportography, and all showed filling of collaterals as an indication of portal hypertension. Out of the 21 patients, 10 later had up to 4 postoperative costal venographies.

Four patients with portal hypertension of extrahepatic origin, demonstrated by percutaneous splenoportography, were investigated preoperatively by costal azygography.

Fifteen patients formerly treated for portal hypertension with portosystemic shunt operation were only investigated postoperatively, 183 to 2,564 days after the operation. In 1 of these cases the patency of the anastomosis was dubious, and 1 was definitely occluded. In the remaining 13, the shunt was patent by clinical standards, and in 3 cases the patency was further confirmed by splenoportography.

A mixed group of 15 patients comprised those with cirrhosis, hepatic metastases, or unclassified severe liver damage, but without signs of portal hypertension. Two of these patients also showed signs of cardiac decompensation. Owing to the uncertain nature of the changes, they are not suited for an evaluation of the venous flow, so that the azygograms of these patients will not be discussed further. The same applies to 2 patients with cardiac disease who had previously been decompensated, although at the time of the investigation there were no signs of cardiac insufficiency. Lastly, 1 patient with a right-sided pulmonary mass of uncertain nature will be left out.

Two patients with cardiac decompensation and 5 with malignant tumors of the right lung will be briefly described below.

A completely normal material cannot be submitted, but 15 of our patients had diseases which should not affect the venous return through the azygos system. In particular, they had no clinical, laboratory, or roentgenologic signs of cirrhosis, portal hypertension, pulmonary or mediastinal disease, or cardiac decompensation.

These groups are listed in Table II, and

TABLE II
CLINICAL CLASSIFICATION

Diagnosis	No. of Patients
Confirmed hepatic cirrhosis with confirmed portal hypertension	21
Extrahepatic portal hypertension	4
Postoperative investigation of patients operated upon for portal hypertension	15
Various hepatic diseases without confirmed portal hypertension	15
Decompensated heart disease	2
Tumor of right lung	5
Miscellaneous	3
No signs of portal hypertension, cardiac decompensation, or pulmonary-mediastinal diseases	15

below the roentgenologic findings will be classified for each group separately.

ROENTGENOLOGIC CLASSIFICATION

A consistent classification of the roentgenologic findings is impossible, as there are even transitions from an obviously "normal" roentgenogram showing 1 or 2 slender intercostal veins and moderate opacification of the azygos vein to "highly abnormal" patterns demonstrating 3-5 intercostal veins and other collaterals of varying caliber, major or minor parts of the internal vertebral venous plexus, and decreased or increased opacification of the azygos vein. The following classification will be used:

1. *Normal Azygograms.* As "normal" we describe venograms showing the appearances which Schobinger calls normal: only 1 or 2 slender intercostal veins and moderate opacification of the azygos vein (Fig. 1, *A* and *B*).

2. *Slightly Divergent Azygograms.* Transitional cases with faint opacification of the azygos vein and possibly filling of small collaterals of doubtful pathologic significance.

3. *Highly Abnormal Azygograms.* In the case of definitely abnormal pictures, showing a varying number of collaterals, it was

necessary to distinguish between those showing an increased opacification of the azygos vein indicating a reduced flow and, according to Schobinger, characteristic of cardiac decompensation or a Valsalva effect, which will be called "highly abnormal, cardiac type," and those with non-filling or incomplete visualization of the azygos vein indicating increased flow, and according to Schobinger, typical of all cases of portal hypertension, which will be called "highly abnormal, portal type" (Fig. 2, *A* and *B*; and 3, *A* and *B*).

RESULTS

A classification of the azygograms from patients with confirmed portal hypertension of intra- as well as extrahepatic origin (Table II, Groups 1 and 2) according to the above principles gives the distribution shown in the first part of Table III. It will be seen that 4 azygograms were completely normal, while another 8 differed only negligibly from the "normal appearances." Among 12 venograms classified as highly abnormal, only 8 showed the appearances characteristic of portal hypertension, while 4 showed an opacification of the azygos system so intense that the findings were more indicative of a reduced flow. None of these patients showed any signs of heart disease. Five had varying degrees of ascites. Three of these patients had azygograms classified as "slightly divergent," one had an abnormal azygogram with signs of increased flow (portal type), while the last patient was a variant beyond classification. None of the other patients had ascites.

As already mentioned, we are unable to submit a completely normal control series, as we have not studied healthy persons by this method. However, it is expressly stated by Schobinger that a liver disease unaccompanied by portal hypertension at the time of the investigation does not affect the azygogram; indeed, this is a presupposition of its applicability. By a critical review of the series in question, we found 15 patients in whom we feel able to

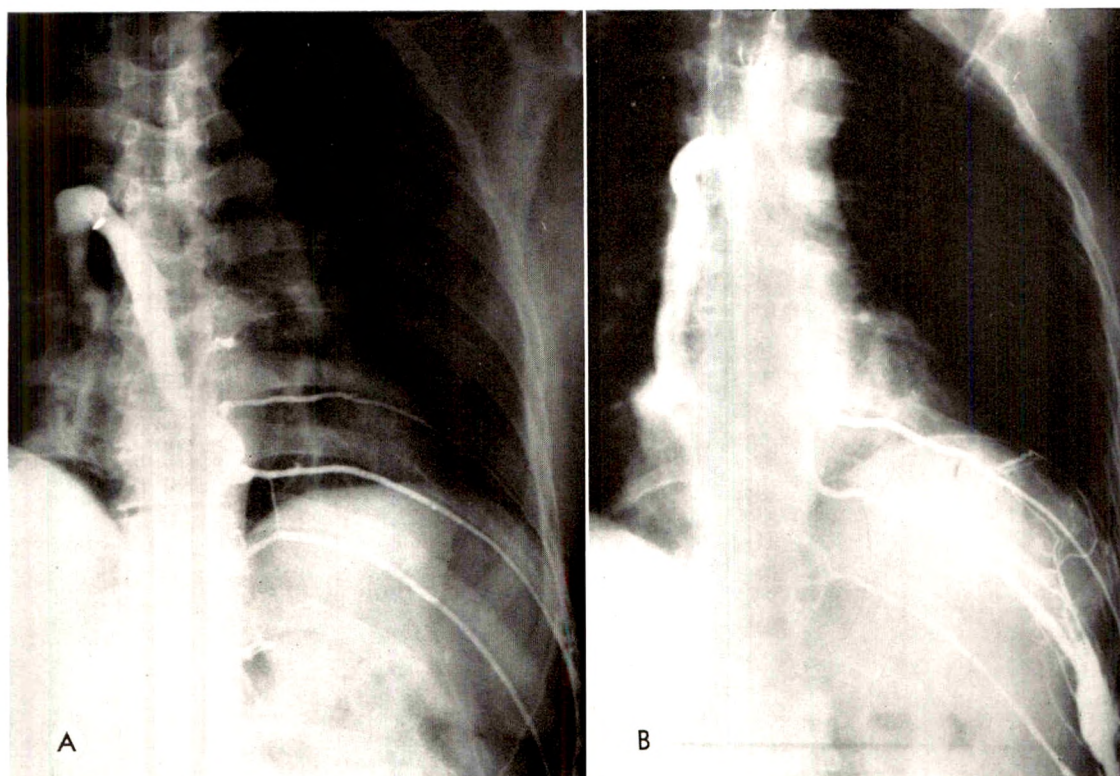


FIG. 3. Abnormal azygograms, reduced flow. (A) Forty-seven year old male without clinical suspicion of portal hypertension or heart disease. (B) Sixty-four year old female with confirmed portal hypertension.

rule out portal hypertension as well as heart disease and mediastinal changes. Probably, a larger number of the patients actually belong to this group, but they were left out because the data were insufficient or the results of the investigations equivocal. The second part of Table

III shows the roentgenographic findings in these patients all of whom ought to have "normal" azygograms. Only 2 showed the simple "normal" venogram. Another 2 venograms differed but little from normal, while 11 were highly abnormal, in most cases of the "portal type" with nonfilling or

TABLE III
ROENTGENOLOGIC CLASSIFICATION

Clinical Group		Azygogram				Others
		"Normal"	"Slightly Divergent"	"Highly Abnormal" (cardiac type)	"Highly Abnormal" (portal type)	
Confirmed portal hypertension		4	8	4	8	1
Normal		2	2	2	9	
Postoperative investigations	with functioning anastomosis	2	3	3	5	
	with nonfunctioning anastomosis				2	

incomplete visualization of the azygos vein and ample filling of collaterals, as a rule including the internal vertebral venous plexus.

Among the patients with confirmed portal hypertension, particular interest was attached to the 12 who were investigated preoperatively as well as postoperatively. Ten had confirmed cirrhosis, while 2 had thrombosis of the splenic vein. In all these patients the function of the artificial shunt was clinically satisfactory throughout the period of the study. In 4 instances splenoportography showed the shunt to be open. These 12 patients had a total of 31 investigations, *i.e.*, 1-2 preoperative and 1-3 postoperative (Table IV). The interval from operation to the postoperative investigation ranged from 10 to 415 days, average 102 days.

Classified according to the above principles, there was preoperatively 1 normal venogram which was unchanged on 2 postoperative investigations. "Slightly divergent" preoperative venograms were found in 3 patients; after the operation they remained unchanged in 2, while 1 became highly abnormal, of the portal type. Lastly, 8 patients had highly abnormal venograms preoperatively; after the operation the appearances were unchanged in 6, normalized in 1 and "slightly divergent" in 1.

Fifteen patients with portosystemic shunts established prior to the commencement of the present study had intra-osseous costal venography 6-84 months after the operation. One had 2 postoperative investigations at an interval of 10 months. Clinically, the shunt functioned satisfactorily in 13 of these patients, while in 2 the function was doubtful. From the third part of Table III, it is apparent that 8 of the patients with a functioning anastomosis showed highly abnormal appearances, although in these cases there had been ample time for normalization. Only 2 patients had completely normal azygograms, while in 3 the azygograms were "slightly divergent." Both patients

with nonfunctioning anastomoses had highly abnormal azygograms.

Finally, the material includes 2 patients with cardiac decompensation, 1 of whom showed the appearances described as typical of this condition, while the other had a normal azygogram. Furthermore, 5 patients had a malignant tumor in the right lung or mediastinum. Four of these patients showed changes characteristic of the superior vena cava syndrome, all the contrast medium passing caudad through collaterals to the inferior vena cava. In 1 of these cases, contrast medium was also injected into a vein of the right arm, visualizing the occluded superior vena cava. The 5th patient had a highly abnormal venogram of the portal type. These results will not be commented on further, but it may be mentioned that several authors, most recently Skinner, Dreyfuss and Nardi,¹² feel that costal venography is a valuable aid in the preoperative assessment of the operability of a lung tumor.

DISCUSSION

As there is a need for a simplified roentgenologic method for demonstrating portal hypertension, the reports on the applicability of costal intra-osseous venography were encouraging. It afforded a method which, although it did not give as full data as splenoportography regarding the cause of portal hypertension, was less of a strain to the patient, technically easy, did not require special equipment, and was considered devoid of risk.

The use of repeated costal intra-osseous venographies to verify the patency of an artificially created shunt, especially in splenectomized patients, was of particular interest.

The applicability of such a method presupposes that the technique can be reproduced, *i.e.*, that the roentgenographic results are not uncontrollably affected by reasonable, minor variations in external factors, such as the choice of the injection site, the speed of injection, and the phase of respiration. In our experience, these de-

TABLE IV

CLASSIFICATION OF PRE- AND POSTOPERATIVE INVESTIGATIONS OF THE SAME PATIENT*

	Venography/Operation	Azygogram			
		Normal	Slightly Divergent	Highly Abnormal (cardiac type)	Highly Abnormal (portal type)
Cirrhosis with portal hypertension	7- 6-1961 Venography	x			
	1-23-1962 Venography	x			
	2-13-1962 Operation				
	4- 5-1962 Venography	x			
	1- 3-1961 Venography				x
	1-17-1961 Operation				
	1-27-1961 Venography				x
	7- 3-1961 Venography				x
	1- 5-1961 Venography				x
	1- 7-1961 Operation				
	1-23-1961 Venography				x
	5-26-1961 Venography				x
	2-27-1962 Venography				x
	3-15-1961 Venography			x	
	3-25-1961 Operation				
	4-12-1961 Venography	x			
	4-17-1961 Venography		x		
	4-27-1961 Operation				
	9- 7-1961 Venography		x	†	
	5-15-1961 Venography				x
Extrahepatic portal hypertension	6- 8-1961 Operation				
	12-19-1961 Venography				x
	5-19-1961 Venography				x
	5-27-1961 Operation				
	11-28-1961 Venography				x
	7-20-1961 Venography		x		
	7-29-1961 Operation				
	11-20-1961 Venography	†	x		
	8-21-1961 Venography		x		
	9-20-1961 Operation				
	10-9-1961 Venography				x
	11-14-1961 Venography		†		x
	11-23-1961 Operation				
	12- 7-1961 Venography				x
	3- 1-1962 Venography			†	x
	11- 9-1961 Venography				x
	11-19-1961 Operation				
	12- 7-1961 Venography				x
	11-28-1961 Venography				x
	12- 1-1961 Venography				x
	12- 5-1961 Operation				
	12-18-1961 Venography		x		
	4-17-1962 Venography		x		

* Ten patients with cirrhosis and portal hypertension confirmed preoperatively, 2 with extrahepatic portal hypertension.

† Classification doubtful; the result should perhaps be listed in this category.

mands seem to be fulfilled, insofar as costal azygograms have proved reproducible in the same patient, both in sequences of immediately repeated injections of contrast medium, with a varying speed of injection, and on injections at different times. Exposure during shallow respiration and during short lasting apnea showed uniform venographic appearances. A Valsalva maneuver seems to entail primarily an increased distention of the veins, but may also lead to filling of accessory collaterals.

Other presuppositions for employing the method are a uniform vascular pattern from patient to patient, well defined changes in the event of portal hypertension, and reversibility of these changes following successful portosystemic shunt operation.

These demands were not fulfilled in our studies (Fig. 1 through 3, inclusive). The azygograms showed all variations from normal to highly abnormal patterns in patients who, judging by clinical findings, ought to have a normal venous return, in patients with confirmed portal hypertension, and in patients investigated a varying length of time after successful portosystemic shunt operation. Our results appear to show that the normal vascular pattern is considerably more variable than described by Schobinger and his associates.

The frequent variations in the normal anatomy of the azygos system of veins further complicate the assessment.

Although the first reports on costal intra-osseous venography appeared as early as 1954, there are only a few reports on the applicability of the method in assessing portal hypertension apart from those published by Schobinger and co-workers. In a study on costal intra-osseous venography in mediastinal tumors Schwartz, Handel and Candel¹¹ described the normal azygogram which they found—in accordance with our experience—more variable than described by Schobinger. In a similar study, Fry, Singer and Tobin³ mentioned 2 patients with portal hypertension, both of whom had normal venograms.

Better conformity with Schobinger's observations was reported by Leger, Montete and Marchand⁴ on the basis of an investigation of "more than 40 patients," 25 of whom had portal hypertension, but they found the filling of collaterals to be extremely variable. In their opinion, nonvisualization of the azygos vein is decisive in diagnosing portal hypertension. Their technique, however, differs considerably from Schobinger's, since they employ a rate of injection of about 30 seconds as compared with Schobinger's 5 seconds or less. Therefore, the results are not directly comparable.

Barlas, Ergun, Oncel and Dinc¹ studied 43 patients, including 20 with cirrhosis, but gave no further data regarding the presence of portal hypertension. Nevertheless, they found the procedure of value in the diagnosis of portal hypertension and described the presence of dilated and tortuous intercostal veins as well as a varying number of collaterals. Unlike Leger, Montete and Marchand⁴ and Schobinger, they found nonvisualization of the azygos vein in only 6 out of the 20 patients. Three of the patients with cirrhosis had normal azygograms.

Owing to the considerable variability in the normal costal azygogram and the partially nonspecific changes observed in patients with confirmed portal hypertension, we do not find that the method, in its pretest form, is suited for demonstrating portal hypertension or for checking the patency of an artificially created shunt.

SUMMARY

Costal intra-osseous venography was carried out 102 times on 80 patients, and particular interest was devoted to the venous return in portal hypertension. The investigations were done by the technique of Schobinger and comprised both pre- and postoperative studies on the same patients and late postoperative studies on a series of patients in whom a portosystemic shunt

had been established previously. A total of 40 patients with present or past portal hypertension were studied.

There was no consistent alteration of the azygogram in the presence of portal hypertension, while pronounced variations in venous pattern were found among normal subjects.

Accordingly, the method is not considered suitable for demonstrating or following the course of portal hypertension.

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ARTERIOGRAPHY OF THE PANCREAS*

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ARTERIOGRAPHY is an important method of examination, not only for visualization of the arteries themselves, but also in the study of parenchymatous organs. Cerebral and renal arteriography has found wide application, and recently arteriography of the liver and spleen has been introduced into practice. Experience with arteriography of the pancreas, however, has hitherto been quite limited. Kincaid and Davis,³ Lewitan *et al.*,⁴ Rigler and Olfelt¹⁰ and others, in discussing translumbar aortography, pointed out the possibility of applying it for diagnosis of pancreatic tumors. Nevertheless, the truly significant advances in arteriography of the pancreas have been made only by *selective* methods of arterial catheterization. Tillander¹³ was one of the first to use selective arteriography for this purpose, and Ödman⁸ outlined the possibilities in selective arteriography of the celiac artery. Olsson^{6,7} and Boijesen² contributed greatly to further development of pancreatic arteriography, introducing a new method of examination and decisively showing the importance of arteriography in the diagnosis of neoplastic processes.

In this report our own experience with arteriography of the pancreas over a period of 4 years is described. Altogether, 91 patients with various diseases of the pancreas were examined (Table I). As a control group, there were 50 arteriographic examinations in patients who had no clinical symptoms of pancreatic disease, and a normal pancreas was surgically confirmed in 12 of these.

ANATOMY

The blood supply of the pancreas is a fairly rich one. However, there is no independent main pancreatic artery; instead, the pancreas receives its blood supply from the arteries of neighboring organs, mainly

TABLE I

Disease	No. of Patients	No. of Operative Confirmations
Pancreatitis	42	18
Pancreaticolithiasis	3	—
Penetration of gastric ulcer into pancreas	3	3
Pseudocyst	8	8
Hyperinsulinism	6	3
Malignant pancreatic tumors	17	15
Gastric carcinoma invading pancreas	12	12
Totals	91	59

from the gastroduodenal artery, the splenic artery, and occasionally the hepatic artery. The superior mesenteric artery also contributes a portion of the pancreatic arterial supply. Some of the supplying branches are constant ones; others vary greatly (Fig. 1).

The head of the pancreas is principally supplied by the pancreaticoduodenal arteries, namely, the superior anterior and the superior posterior ones, which branch off from the gastroduodenal artery, and the inferior anterior and inferior posterior ones, given off by the superior mesenteric artery. These generally have a diameter of approximately 2 mm., they anastomose richly, and the anastomoses form arterial arches on the anterior and posterior surfaces of the head of the pancreas. From these arteries, relatively long branches supply the duodenum, and shorter ones enter the pancreas.

In the region of the pancreatic neck, there lies the dorsal pancreatic artery. It arises most frequently from the first portion of the splenic artery, but it may also be a branch of the celiac artery, the hepatic artery, or the superior mesenteric artery. Occasionally, it is completely absent. The dor-

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sal pancreatic artery is usually 1.5–2.0 mm. wide. It divides after a short course into two branches, the right branch and the left one. Its right branch anastomoses with arteries on the anterior surface of the head of the pancreas; its left branch leads to the body of the pancreas, where it communicates with the inferior pancreatic artery and occasionally is actually the origin of the latter.

The body and tail of the pancreas receive their blood supply mainly from the splenic artery, which gives rise to the arteria pancreatica magna, the caudal pancreatic artery, and several smaller branches—the rami pancreatici. Also, the superior mesenteric artery provides a small (and sometimes a rather large) arterial supply to the body of the pancreas. The arteria pancreatica magna arises from the splenic artery to the left of the spine. It is short and approximately 2.0–3.0 mm. wide. It enters the pancreatic parenchyma and ramifies richly in the region of the body and the tail. The caudal pancreatic artery originates from the peripheral part of the splenic artery, or from its hilar branches, and anastomoses with the other branches. It is narrow and is occasionally replaced by several smaller vessels, or it may be entirely absent.

The inferior pancreatic artery is found near the lower margin and posterior surface of the body and tail of the pancreas. It usually is 1.0–1.5 mm. in diameter and its ramifications are highly variable. Sometimes, it originates from the dorsal pancreatic artery, at other times from the superior mesenteric artery, and occasionally from lesser branches in the head of the pancreas.

TECHNIQUE

We perform pancreatic arteriography by percutaneous catheterization after the method of Seldinger, choosing usually the femoral route. Selective arteriography of the celiac artery and the superior mesenteric artery is utilized, the injections being made either consecutively with one catheter

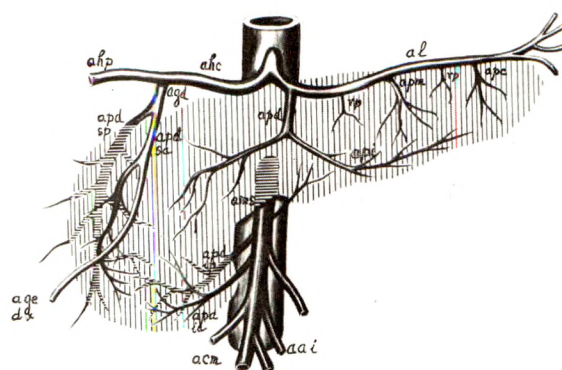


FIG. 1. Anatomy of the arterial blood supply of the pancreas: ac—celiac artery, al—splenic artery, ahc—common hepatic artery, ahp—hepatic artery proper, agd—gastroduodenal artery, apdsp—superior posterior pancreaticoduodenal artery, apdsa—superior anterior pancreaticoduodenal artery, agedx—right gastroepiploic artery, apd—dorsal pancreatic artery, apm—arteria pancreatica magna, apc—caudal pancreatic artery, rp—rami pancreatici, api—inferior pancreatic artery, ams—superior mesenteric artery, apdip—inferior posterior pancreaticoduodenal artery, apdia—inferior anterior pancreaticoduodenal artery, acm—middle colic artery, aai—intestinal arteries.

ter or simultaneously with two catheters, as suggested by Olsson⁷ and Boijsen.² For the simultaneous approach, one catheter is introduced through the right femoral artery and the other through the left femoral artery. In those patients in whom it is not possible to introduce a catheter selectively into the celiac artery or the superior mesenteric artery, high abdominal aortography is used; in our series, this was necessary in only 5 patients.

The catheter used for selective catheterization is the green Ödman-Ledin catheter (Kifa), the end of which is bent into a small 90° to 180° curve. Under fluoroscopic control, the catheter is passed to the level of the diaphragm. After removal of the spring guide, the catheter is rotated so that its curved end is directed ventrally. The catheter is then slowly retracted to the approximate level of T12 or L1, where the tip enters the celiac artery either spontaneously or after further manipulation. Correct position of the catheter tip is confirmed by a test injection of 3–5 cc. of contrast agent. Selective catheterization of the superior

mesenteric artery is accomplished similarly, the origin of this artery being 1.0–3.0 cm. caudal to the root of the celiac artery.

As contrast medium, we use 76 per cent Uromiro (Bracco) or 76 per cent Urografin (Schering). Celiac arteriography is performed with 30–40 cc., while superior mesenteric arteriography requires 20–25 cc. For simultaneous arteriography, 50–60 cc. is used. Injection of the contrast agent is accomplished with a Gidlund syringe under a pressure of 4 atmospheres. An Elema seriograph is utilized for roentgenography, the first 6 exposures being at half second intervals, the next 2 or 3 exposures at one second intervals, and the final 6 exposures at three second intervals. Curix-Rapid Gevaert film is used, with exposure factors of 100–115 kv., 350 ma., and 0.1 second. To improve visualization of the pancreatic vessels, the stomach is sometimes insufflated with an effervescent powder.

NORMAL APPEARANCES

Selective arteriography of the pancreas has two stages: the vascular stage (Fig. 2) and the parenchymatous stage. Both stages (or phases) are subject to considerable variability, for which allowance must be made in the interpretation of normal anatomy.

During the vascular phase, there is variable filling of arteries in the vicinity of the



FIG. 2. Normal picture of simultaneous arteriography of celiac and superior mesenteric arteries, with insufflation of stomach.

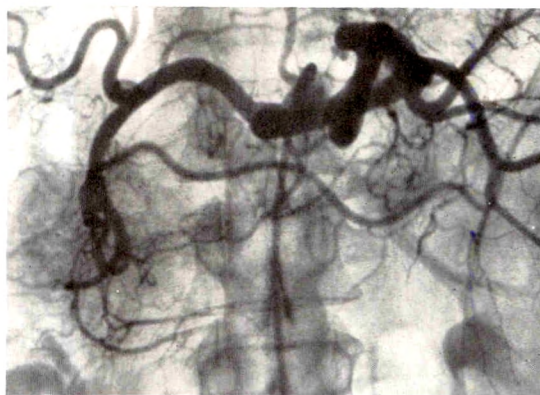


FIG. 3. Arteriography of the celiac artery in hepatitis and splenomegaly, with well demonstrated vessels of the entire pancreas. The tail of the pancreas is displaced by an enlarged spleen.

pancreas. The branches of the head—the pancreaticoduodenal arteries—are usually well-shown, especially during simultaneous arteriography. In consecutive arteriography, only the branches arising from the injected artery are ordinarily filled. However, it occasionally happens that branches of the artery which were not injected may be opacified through anastomoses (Fig. 3) in which case the entire region of the head may be adequately visualized. In addition, the vascular supply arising from the splenic artery is generally well filled, especially the arteria pancreatica magna. The latter usually courses obliquely to the left side, promptly dividing into many smaller radicals. The dorsal pancreatic artery is not constantly visualized, but in those cases where it does opacify it is easily identified by its typical ramification in the shape of an inverted T. The caudal pancreatic artery and the rami pancreatici are sometimes visible as fine branches communicating with surrounding arteries. The inferior pancreatic artery is fairly often seen, and it is relatively long. It is important to keep in mind that vessels within the left portion of the pancreas must be carefully differentiated from superimposed branches of the gastric artery, especially in patients with an elongated stomach.

The parenchymatous phase of pancreatic arteriography is even more variable than

the arterial stage. It commences 6–10 seconds after the beginning of the injection and is usually quite pronounced, especially in simultaneous arteriography or in arteriography of the celiac artery. The pancreas during this phase becomes diffusely opacified, contrasting sharply with its background. However, opacification of the entire pancreas is the exception rather than the rule. This is especially true in the region of the head of the pancreas, where the parenchymatous phase is least marked and sometimes can hardly be discerned. The left portion of the pancreas is usually better opacified, particularly in those cases where the arteria pancreatica magna is of generous size. In the latter case, the body and tail of the pancreas may be readily identified, from the spine up to the hilus of the spleen. During the parenchymatous phase, the splenic vein and the portal vein are also often depicted, and thus their relation to the pancreas may be easily ascertained.

PANCREATITIS

In 42 patients with inflammatory conditions involving the pancreas, we found highly variable pictures which demonstrated a wide scale of findings, ranging from normal appearances to a significant increase in number and deformity of pancreatic branches, or, conversely, a marked reduction in vascular supply. In 6 patients, the arteriographic picture was a normal one; these were patients with secondary pancreatitis, or those in the quiescent stage of chronic relapsing pancreatitis. The most frequent finding, observed in 31 patients, was enlargement of the pancreas associated with irregularity of shape and an increase in arterial blood supply (Fig. 4, *A* and *B*). The parenchymatous phase was accentuated, and arterial deformation occasionally could be seen. These findings were of variable degree and corresponded on a whole with the character and stage of the disease process.

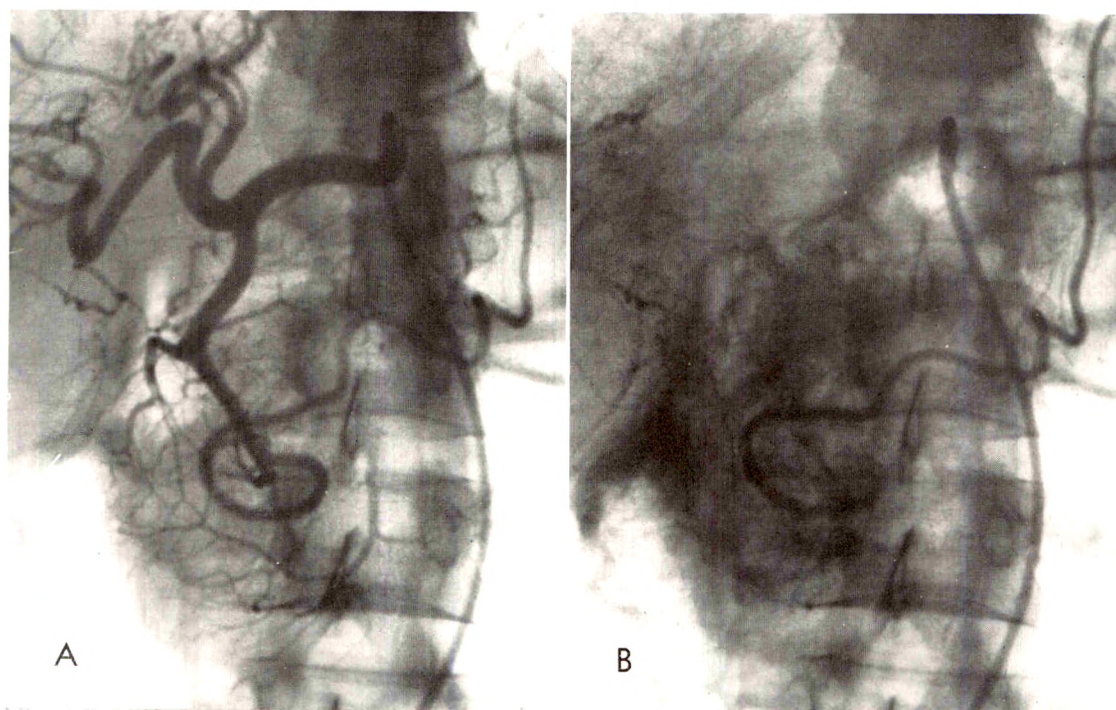


FIG. 4. Subacute pancreatitis, with enlargement and hypervascularity of the right side of the pancreas. Arteriography of the common hepatic artery. (*A*) Arterial phase; (*B*) parenchymatous phase.

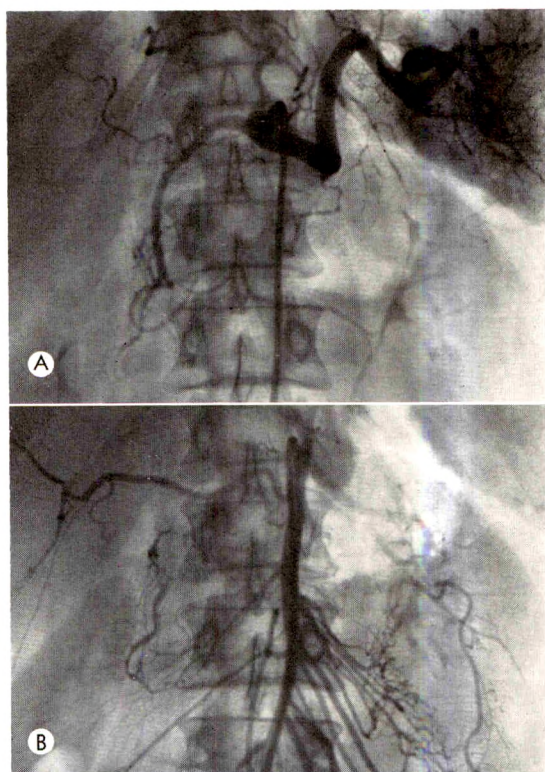


FIG. 5. Subacute pancreatitis, with involvement of the entire pancreas, but especially of its left side in which the branches are disfigured. (A) Arteriography of celiac artery; (B) arteriography of superior mesenteric artery.

In patients examined during the relapsing stage of chronic pancreatitis, or soon after a more acute phase, we observed slight pressure deformity of the surrounding arteries, and the arterial supply of the pancreas appeared to occupy a larger area than normal. In a well pronounced parenchymatous phase, enlargement of the pancreas was usually still more apparent; its shape appeared irregular, the contours were often knobby, and the neighboring veins were occasionally found to be compressed. These alterations were frequently well localized and affected only the head of the pancreas, or its left portion (Fig. 5, A and B), less frequently the entire pancreas was affected. This was the picture observed, for example, in a female patient following splenectomy. However, in her case it could not be determined whether inflammatory

hypervascularity accounted for the findings, or whether the excessive filling of pancreatic vessels was due to a large quantity of contrast agent having been delivered by a functioning remnant of the splenic artery.

In patients with a less advanced stage of pancreatitis, inflammatory hypervascularity was not so pronounced. The parenchymatous phase, however, often revealed irregular knobby contours of the pancreas, and occasionally the pancreatic outlines were also rather blurred. In several cases, the arteries were variously disfigured within the enlarged pancreas; usually, they appeared straightened and even rigid, but now and then they appeared irregularly narrowed with roughened outlines (Fig. 5, A and B).

In the remaining 5 patients with advanced forms of pancreatitis, mostly of the atrophic type, the arterial supply was found to be diminished or barely visible and the parenchymatous phase was practically absent.

PANCREATICOLITHIASIS

All 3 of these patients had concretions within the left portion of the pancreas. Arteriographic findings were virtually identical in the 3 cases. The left portion of the pancreas appeared very poorly vascularized and in one patient the vascular supply was totally invisible. This portion of the pancreas displayed practically no parenchymatous phase. In contrast, the head showed normal or increased vascularity, and the parenchymatous phase demonstrated it to be of normal shape although slightly enlarged. In one patient, during the parenchymatous phase, occlusion of the splenic vein was incidentally visualized.

PENETRATION OF GASTRIC ULCER INTO THE PANCREAS

In cases of gastric ulcer penetrating into the pancreas, changes were observed which were less marked but otherwise similar to those in pancreatitis, consisting mainly of hypervascularity. The hypervascularity in these cases, however, was localized and ap-

peared well-developed only in the immediate vicinity of penetration. In one patient with a prepyloric gastric ulcer, there was increased vascularity in the region of the head of the pancreas; in those patients with ulcer of the posterior wall of the body of the stomach, the hypervascularity was found within the left portion of the pancreas. The uninvolved portions of the pancreas showed no alterations from normal.

PSEUDOCYST OF THE PANCREAS

Of 8 patients with postnecrotic pseudocysts, 4 were examined preoperatively and 4 were studied postoperatively.

In 2 of the preoperative patients, the pseudocyst originated from the head of the pancreas, in another from the body of the pancreas, and, in the fourth preoperative patient, there was one pseudocyst in the head and another within the body of the pancreas. Arteriography showed characteristic pressure changes involving the surrounding vessels in all of these patients. At the site of the cyst, there was an absence of the vessels. Diminished vascularity and deformity of vessels could be seen in the remaining parts of the pancreas, with a faint or inadequate parenchymatous phase and occlusion of the splenoportal trunk with collateral circulation. Displacement of surrounding vessels was in the direction of growth of the pseudocyst. In one instance, a pseudocyst of the head of the pancreas displaced the common hepatic and gastroduodenal arteries to the right; in a second case, the gastroduodenal artery was pushed to the left and its pancreaticoduodenal branches encircled the pseudocyst (Fig. 6). In a third patient, it was principally the inferior pancreaticoduodenal arteries which were compressed. The pseudocyst of the body of the pancreas in one of the patients displaced the superior mesenteric artery to the right.

Four postoperative patients were examined after emptying of their cysts. In a female patient, examined 2 months following acute necrosis and sequestration of the tail of the pancreas, arteriography demon-

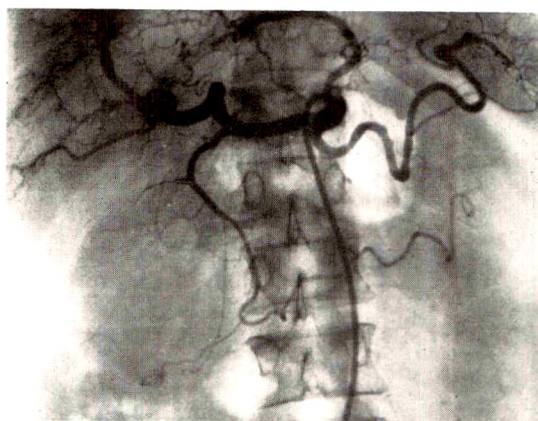


FIG. 6. Large pseudocyst of the head and smaller pseudocyst of the body of the pancreas, with pressure changes on surrounding vessels. Arteriography of celiac artery.

strated considerable hypervascularity and enlargement of the head of the pancreas, with a well marked parenchymatous phase. The left portion of this particular patient's pancreas appeared quite avascular. The remaining postoperative patients, examined 2 or 3 years following surgery, showed diminished ramification of pancreatic vessels, deformity of the arteries, and an impaired parenchymatous phase.

HYPERINSULINISM

Our series included 6 patients with various forms of hyperinsulinism. In 3 patients, arteriography disclosed a tumor arising from Langerhans' cells (insulinoma); in 2 patients, diffuse hypertrophy of the left portion of the pancreas was found; and, in 1 patient with occasional attacks of hyperinsulinism, the arteriographic findings were within normal limits.

The picture of insulinoma was quite characteristic in 2 of the patients. In the case of a 12 year old boy with an insulinoma the size of a cherry in the tail of the pancreas, a rich vascular network was demonstrated within the tumor during the arterial phase (Fig. 7A); during the parenchymatous phase, the entire tumor became diffusely opacified and contrasted sharply with its surroundings (Fig. 7B). In a 28 year old patient with an insulinoma, there had been a left-sided pancreatectomy followed by to-

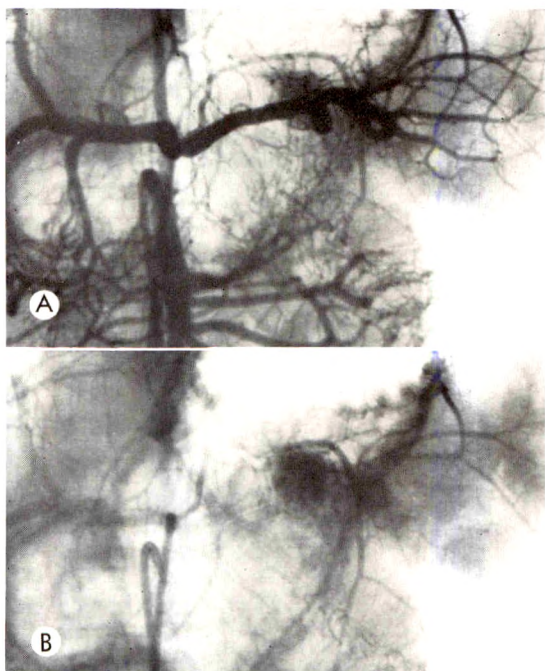


FIG. 7. Insulinoma of the tail of the pancreas, the size of a cherry. Simultaneous arteriography of celiac and superior mesenteric arteries, with insufflation of the stomach. (A) Arterial phase; (B) parenchymatous phase.

tal pancreatectomy, 1 year before the examination. Soon after these operations, hyperinsulinism had reappeared. Arteriography of the celiac artery revealed typically staining tumor foci within the lower part of the right lobe of the liver. One of these was the size of a nut, the other the size of a lentil (Fig. 8). Our findings were confirmed at surgery and, following resection of the involved portion of the liver, the patient has remained free of complaints. In a third patient, not yet operated upon, arteriography demonstrated 2 small areas of neovascularity within the body of the pancreas.

In the patients with diffuse hypertrophy of the left portion of the pancreas, the arterial ramifications were found to be markedly increased in the body and within the tail, but no significant arterial deformity was observed. The parenchymatous phase was well developed in these areas, demonstrating enlargement of the left portion of the pancreas and uneven contours. No neovascularity was found.

MALIGNANT TUMORS OF THE PANCREAS

There were 17 patients examined in this category, 14 with carcinoma, 1 with a cystic sarcoma, 1 with an angiosarcoma, and a woman with a nesidioblastoma and metastases to the liver. In 6 patients, the tumor arose within the head of the pancreas; in 8 patients, it involved the head and body of the pancreas; and, in 3 patients, the body and tail of the pancreas were involved.

The patients with carcinoma and the patient with cystic sarcoma showed similar pictures. In these cases, the principal finding was neoplastic invasion of vessels in the vicinity of the tumor. This was found in all cases, although its extent was variable. Occasionally, only a localized segment of an artery showed rigidity and unsharpness of contours. In such cases, the findings were difficult to evaluate, especially when smaller vessels were affected. In other cases, the findings were quite convincing, with the in-

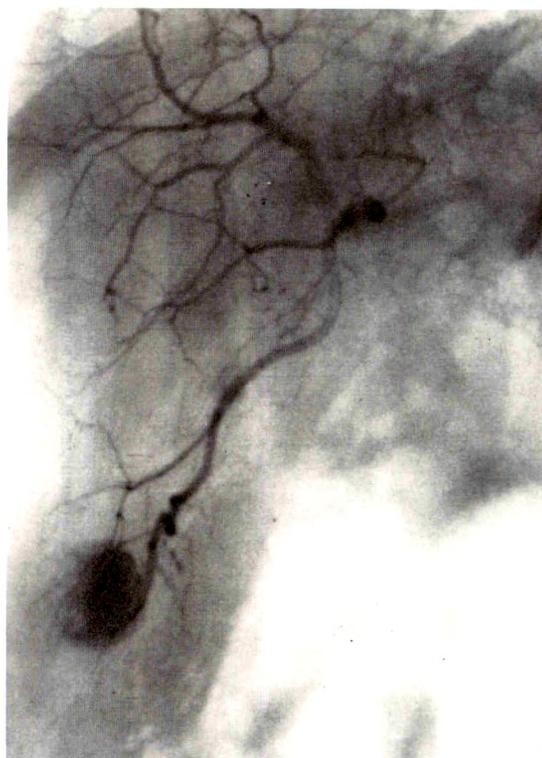


FIG. 8. Two tumor foci of an insulinoma in the lower part of the right lobe of the liver. Hepatic arteriography.

vaded vessel being obviously stenosed, rigid, and of jagged contours (Fig. 9, *A* and *B*). Beyond the area of vascular stenosis, slight dilatation of involved vessels could be seen. In tumors involving the head of the pancreas, the gastroduodenal artery and its branches were primarily affected, with less frequent involvement of the superior mesenteric and common hepatic arteries. In 1 patient (Fig. 11), a small carcinoma encircling the trunk of the superior mesenteric artery produced such diffuse changes that they were mistaken for arteriosclerotic alterations. Neoplasms involving the body of the pancreas tended to infiltrate the central portions of the common hepatic and splenic arteries, and occasionally the left gastric artery (Fig. 10). Lesions arising in the body and tail were characterized mainly by their effects upon the splenic artery. The pancre-

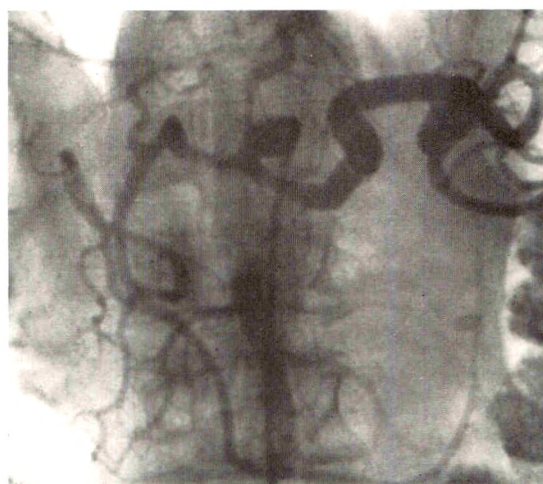


FIG. 10. Carcinoma of the body and head of the pancreas, with encroachment upon central portions of the common hepatic, splenic and left gastric arteries. Arteriography of celiac artery, with filling of superior mesenteric artery through anastomoses in the region of the head of the pancreas.

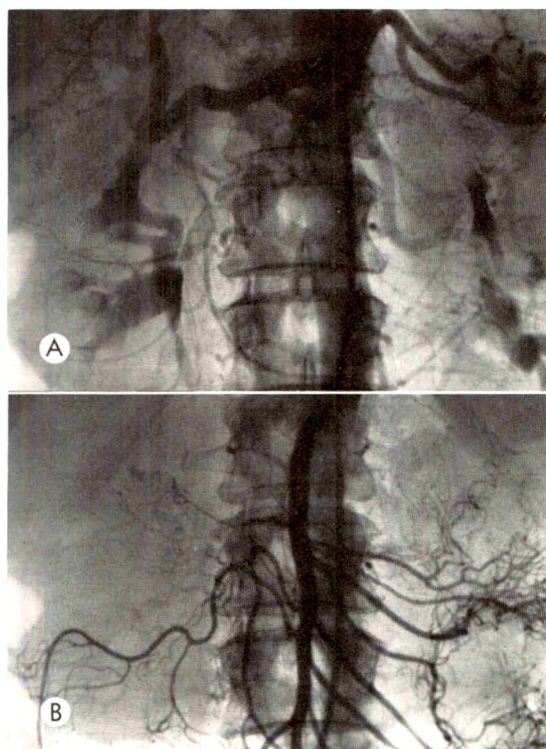


FIG. 9. Carcinoma of the head of the pancreas. (*A*) Arteriography of the celiac artery demonstrates invasion of gastroduodenal artery, with pressure changes on the pancreaticoduodenal arteries; (*B*) arteriography of the superior mesenteric artery reveals hypervascularity in the region of the tumor.

atic branches in the region of the body and tail were only occasionally affected, and for the most part were not visualized.

Pressure changes due to carcinoma were of variable degree, with arteries in the im-

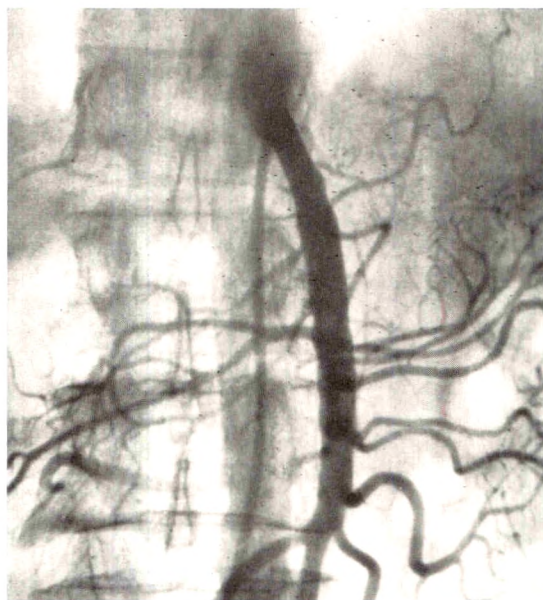


FIG. 11. Carcinoma of the head of the pancreas, encircling the base of the superior mesenteric artery. Arteriography of superior mesenteric artery.

mediate vicinity of the pancreas and the pancreaticoduodenal arteries showing the most marked changes. Neovascularity due to tumor was found in 6 patients. However, in only 2 patients was the finding convincing, and in these cases the neovascularity had the character of fine plexuses; in the other 4 patients, neovascularity was barely discernible and therefore less convincing. The parenchymatous phase was usually not well marked in cases of pancreatic carcinoma. Delayed roentgenograms often revealed occlusion of the splenic vein with an opacified collateral circulation.

In the patient with angiosarcoma and the woman with nesidioblastoma, the findings were in a class by themselves. The angiosarcoma, originating from the body and tail of the pancreas, caused infiltration of the splenic artery. Its principal manifestation, however, was a rich neovascularity. This consisted of irregular plexuses, with numerous anastomoses leading to the portal circulation and to varices in the wall of the fundus of the stomach, as well as wide veins

draining towards the colon (Fig. 12). In the female patient with extensive nesidioblastoma, only abdominal aortography was possible. There was nonvisualization of the splenic artery, obviously due to neoplastic thrombosis. Despite this, many irregular neoplastic vascular plexuses were filled, with venous shunts deriving from them.

GASTRIC TUMOR INVADING THE PANCREAS

The 12 patients examined in this group exhibited a variety of findings. In most cases, the neoplasm arose from the middle or the upper portion of the stomach. In 7 cases, it was manifested by neovascularity, and there were several examples of dense staining during the parenchymatous phase (Fig. 13, *A* and *B*). These findings were especially well demonstrated with simultaneous insufflation of the stomach. There were no instances of gastric neoplasm invading the splenic artery, common hepatic artery, or superior mesenteric artery. Vascularity of the pancreas was normal or slightly increased, especially on the left side. During the parenchymatous phase, the left portion of the pancreas was well opacified and was occasionally found to be depressed, with blurring of its upper margin. In 3 cases, the opacified pancreas blended directly with the shadow of the stained tumor.

DISCUSSION AND CONCLUSIONS

Although previous experience with arteriography of the pancreas is limited, there remains a basis for drawing certain conclusions. To date, since abdominal aortography has failed to provide sufficient information, selective arteriography of the celiac artery and superior mesenteric artery appears to be the method of choice. The main issue is whether to perform selective arteriography of the two arteries by simultaneous injection, or in successive stages. Both of these approaches have their individual advantages and disadvantages. In simultaneous arteriography, the vessels of the head of the pancreas are usually better demonstrated and, also, the parenchymatous phase is

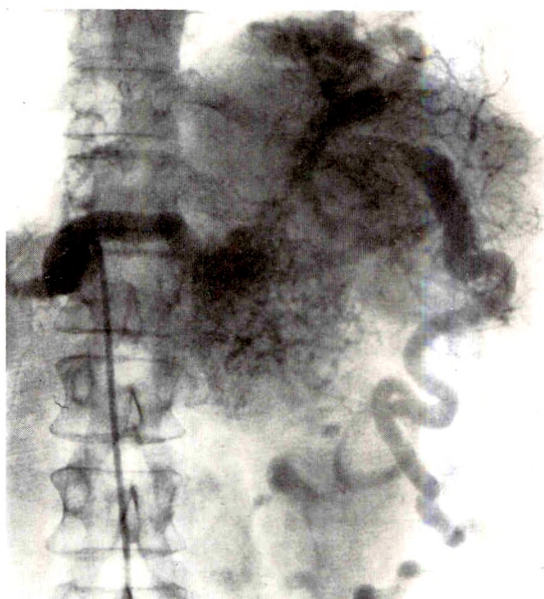


FIG. 12. Angiosarcoma arising in the body and tail of the pancreas, with filling of many newly formed vessels and opacification of venous anastomoses leading to the stomach and colon. Arteriography of celiac artery.

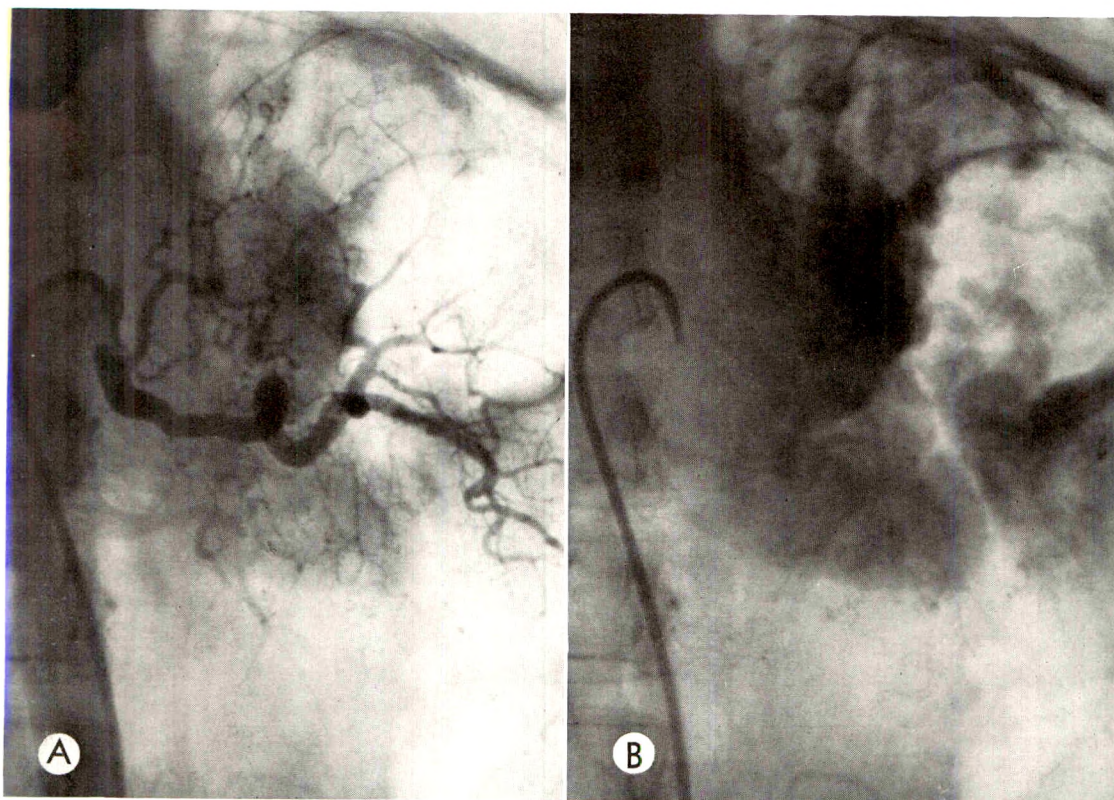


FIG. 13. Gastric tumor invading the body of the pancreas. (A) Arterial phase showing neovascularity in the region of the tumor; (B) parenchymatous phase demonstrating diffuse opacification ("staining") of the tumor and the pancreas.

more marked. This method, however, necessitates puncture of both femoral arteries and thus consumes more time; moreover, the large number of opacified vessels sometimes makes appraisal quite difficult. Successive arteriography, on the other hand, entails more irradiation of the patient while requiring but one needle puncture. It is technically less pretentious, and roentgenograms obtained in succession often present a better survey. All things considered, we find ourselves using successive arteriography more frequently than in the past.

Further improvement in diagnosis will undoubtedly be achieved by application of selective arteriography to particular branches of the celiac and superior mesenteric arteries. For example, with a suspected lesion in the head of the pancreas, the most useful information would be derived from selective arteriography of the gastroduo-

denal artery; by the same token, selective arteriography of the splenic artery would be ideal for evaluation of processes involving the left portion of the pancreas.

Visualization of the pancreas may be improved by insufflation of the stomach. This should be reserved, however, for elongated stomachs where vascular ramifications may obscure the left portion of the pancreas. Roentgenograms obtained this way are, of course, more difficult to reproduce for publication because of the wide range in contrast.

Until now, examinations have been performed with the stomach empty, implying minimal function of the pancreas with correspondingly reduced circulation. A theoretic approach to better demonstration of the pancreas might be to examine it in the actively functioning state. However, Ödman's⁸ experiments with examination fol-

lowing administration of fats and cholecystokinin have so far been unsuccessful. We have tried to influence the examination by secretin, but results to date have been unimpressive.

Evaluation of pancreatic arteriograms must be circumspect, taking into full account the great variability and wide range of normal. It is important to appreciate the distribution of the vessels which encircle the pancreas or are embedded within its parenchyma. Being familiar with these features, the location and size of the pancreas may be estimated and pathologic processes can be discerned at an early stage. It is also important to search carefully for neovascularity, this being of great significance in the diagnosis of insulomata. Not to be underestimated is the parenchymatous phase, which makes possible a most exact determination of size, shape, and position of the pancreas (especially its left portion). Ödman's supposition, namely, that it should be possible to discern even a small cyst during the parenchymatous phase, unfortunately is rarely confirmed in practice. In late phases, it is also important to assess the venous vascular bed, paying particular attention to the splenic and portal veins.

Pancreatic arteriography is not of uniform value in all diseases of the pancreas. Its single most important application is in the diagnosis of hyperinsulinomata and tumors of Langerhans' cells. These tumors, the diagnosis of which has been very difficult in the past, are reliably demonstrated by arteriography. As long as they are of discernible size, their location may be accurately determined and secondary foci in surrounding organs may even be recognized, thus facilitating surgical removal. Moreover, arteriography is valuable for the diagnosis of other types of tumor, both benign and malignant. On the whole, it permits reliable differentiation of such tumors, the criterion being the presence of pressure deformity or invasion of affected vessels. Early diagnosis of malignant tumors is often made possible by pancreatic arteriography. In these instances, it is important to

search mainly for vessel invasion. Unfortunately, involvement limited to the smaller arteries will present a difficult decision since similar alterations may result from inflammatory diseases of the pancreas; in the latter condition, however, a greater number of vessels is usually affected to a more marked degree.

Arteriography further assists the surgeon in deciding upon the operability of neoplasms. If a vessel of vital importance is affected by the infiltrative process (*e.g.*, the superior mesenteric or common hepatic artery), the tumor will already be inoperable. Also, by demonstrating the vascular abnormalities which are so frequent in these areas, pancreatic arteriography will aid the surgeon in avoiding complications. In the inflammatory diseases, arteriography helps to a certain extent in ascertaining the degree of involvement, and thus may assist in planning the operation. Pancreatic arteriography also represents an important method for differential diagnosis of gastric neoplasms and for more precise determination of their dorsal spread.

The diagnostic possibilities of pancreatic arteriography may be appreciated by comparing it with other methods of examination (we intentionally exclude tumors of Langerhans' islets, for which arteriography is the method supreme). Comparison with the usual examinations of the stomach and duodenum is overwhelmingly in favor of arteriography, which many times has demonstrated changes in cases where even careful examination of the stomach and duodenum revealed no abnormality. Especially interesting is a comparison with specialized methods of examination, such as hypotonic duodenography and splenoportography. In our opinion, splenoportography and hypotonic duodenography are more reliable in the diagnosis of pancreatitis than is arteriography, even though these too, have some drawbacks. To some extent, this belief may be due to our more extensive experience and clearer diagnostic criteria with the former methods. In contrast, the diagnosis of neoplastic processes depends principally on

localization and spreading of the tumor. Thus, in one of our patients with cancer of the body of the pancreas spreading anteriorly, pancreatic arteriography presented a clearly pathologic finding, whereas splenoportography and hypotonic duodenography were almost normal. On the other hand, splenoportography was significantly positive in a patient with a tumor of the body and tail of the pancreas, whereas arteriography showed only a questionable infiltration of the pancreatic artery. In the remaining patients, findings by the various methods were generally in agreement. Accordingly, we consider pancreatic arteriography, splenoportography, and hypotonic duodenography to be methods complementing one another. So far as practicable, it would appear advantageous to perform all three procedures in patients with suspected pancreatic disease. At the same time, we do not advocate eliminating the common examinations of the stomach and duodenum; these retain their importance as basic examinations despite introduction of new methods, and are certainly useful for broad orientation about the pancreas.

SUMMARY

Following a survey of the literature and review of the regional vascular anatomy, the authors summarize their personal experience with selective pancreatic arteriography. Arteriograms have been obtained in 91 patients with various diseases of the pancreas, and examinations in 50 patients without evidence of pancreatic disease have been evaluated as controls. The technique of the examination is described, followed by descriptions of the normal pancreatic arteriogram and the pathologic findings in pancreatitis, pseudocysts, hyperinsulinism, malignant pancreatic tumors, and invasion of the pancreas by gastric ulcers and neoplasms. The concluding discussion concerns the technique of examination and

possibilities for its improvement, the principles of evaluating the roentgenographic findings, and a comparison of arteriography with other methods for the diagnosis of pancreatic diseases.

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ARTERIOGRAPHY IN ORTHOPEDICS*

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RAPID developments in the field of arteriography have greatly broadened its applications by its capability to accurately reproduce *in vivo* anatomy and pathology. A wide experience in the interpretation of morbid vascular anatomy has been obtained not only in diseases of the cardiovascular system but also in diseases of the renal parenchyma, the abdominal visceral organs and the brain. The results have shown that these detailed studies have provoked much interest. In several special situations, as carotid and renal angiography, the examinations are indispensable to modern diagnosis and treatment. It was thought that the application of arteriography to various orthopedic problems might result in substantial diagnostic advantages. With this purpose in mind, we sought to build up a background of experience in trauma, neoplastic disease and in the differential diagnosis of inflammatory disease *versus* tumor. The necessity for arteriography in such conditions as congenital hypertrophy of an extremity has already been established.

An evaluation of our experience with osseous arteriography, while admittedly still limited, has indicated the following positive advantages: (1) increases the opportunity for preoperative diagnosis; (2) permits a graphic representation of vascular anatomy; (3) allows confirmation of suspected malignancy or of malignant degeneration of a benign tumor; and (4) aids in determining the optimum site for biopsy of a suspected malignant tumor by demonstrating the area of maximum vascularity. As a result, arteriography has been found to be valuable in the investigation of a variety of diseases involving the musculoskeletal system.

Selective arteriography has been performed in 40 patients with the deliberate

intention of analyzing the vascular structure of a given lesion. Certain of these cases have been chosen for discussion and case presentation because they serve to demonstrate the role that angiography played in either diagnosis or treatment or because the case represents a basic example of some important facet of this radiographic technique.

It is interesting that the first efforts in the field of arteriography were directed toward the differential diagnosis of neoplasms in bone. The work of dos Santos,⁴ Caldas², and Fariñas⁵ actually provided the major impetus to the development of modern selective arteriography. The value of serial roentgenographic examinations was demonstrated by these investigators and, subsequently, improvements in the technique enabling a wider application of angiography were formulated by Radner,¹² Seldinger,¹³ Ödman¹⁰ and others. In conjunction with the improvement of available contrast media and the modernization of roentgenographic equipment, the role of angiography rapidly expanded in medicine.

The technical approach is variable and determined by the specific problem. In its simplest form, peripheral arteriography by direct needle puncture is employed in many patients with lesions of the extremities. This often involves direct puncture of the brachial or common femoral arteries where the use of small gauge needles is preferable to the larger sized needles (greater than 18 gauge) since minor puncture wounds in the artery result, minimizing the aftercare of the patient. The choice of the type of needle is primarily a personal one, varying between arterial needles of the Cournand type, ordinary short beveled needles, or specially designed arterial nee-

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dles with blunt tipped outer cannulas. Arterial needles have been devised with specially designed mandrins to permit the safer passage of the needle through the lumen of the vessel to ensure a well maintained position throughout the examination. Generally 50 per cent diatrizoate contrast agents (or their equivalents) meet the requirements of the examination. The amount of contrast medium varies between 10 and 20 cc., depending upon the size of the part to be examined and the presumed nature of the lesion.

The modified Seldinger technique of catheter replacement arteriography is also actively employed, most often with the femoral artery as the site of entrance into the arterial system. While the retrograde passage of a catheter from this site to the subclavian vessels provides excellent visualization of the subclavian, axillary and brachial arteries, visualization of the vascular supply to the forearm and hand is generally not adequate. Accordingly, when such areas are under study, direct needle puncture of the brachial artery is employed. Lesions of the thigh, knee or the proximal leg can be adequately examined by needle puncture of the femoral artery. The peripheral portions of the lower limb can be demonstrated best by the antegrade passage of a catheter from the femoral into the popliteal artery, employing pressure injection of contrast medium. Precise detail of the peripheral vasculature of the leg and foot is obtained in this fashion. Individual situations may prohibit the use of vessels within the diseased limb, in which case a catheter is passed from the opposite side in a retrograde fashion to the aortic bifurcation and then directed antegrade through the common iliac system of the leg to be examined. These various techniques carry a very minor rate of complications, most of which are insignificant in the hands of experienced personnel. All examinations adhere to certain basic principles: (1) it is desirable to inject the contrast medium into a vessel at a point as close to the lesion as is feasible;

(2) whenever possible superimposition of vessels not specific to the problem should be avoided; and (3) high quality, highly detailed serial roentgenograms covering the arterial, capillary and venous phases of the blood flow through the diseased tissue are preferable.

INTERPRETATION OF STUDIES

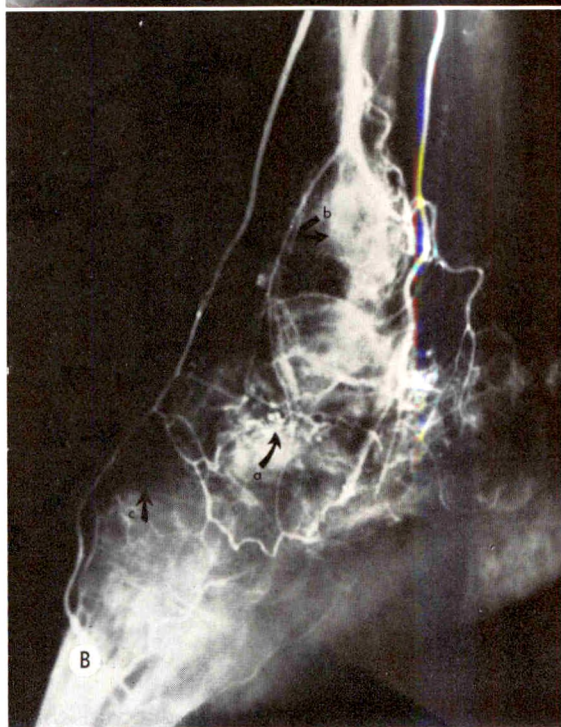
The interpretation of arteriograms is based upon the analysis of the opacified arterial supply and to some extent upon the venous drainage of the lesion; it depends upon an evaluation of the caliber, course, contour and number of vessels as well as a study of the dynamics of flow and its comparison with the known normal. The abnormal condition of "staining" of a lesion is occasionally encountered. This consists of an overall increase in density caused by the accumulation of the contrast agent in innumerable thin walled vascular channels. The individual channels, however, cannot be identified on the roentgenogram and the radiopaque blush persists into the venous phase beyond the period of opacification of the arterial system (Fig. 1, *B* and *C*; and 2, *B* and *C*).

As is shown later, the evaluation of post-traumatic conditions producing disruption, compression or occlusion of arteries as well as aneurysms or fistulae (Fig. 3; and 4, *A* and *B*) poses no diagnostic difficulties and precise information as to size and position of the lesion as well as the state of collateral circulation, etc., can be clearly defined. This information is invaluable to the clinician.

The differential diagnosis between inflammation, benign and malignant tumors is more difficult. These lesions all may produce tumor-like swelling which may cause displacement of vessels, leading to increased vascularity. The morphologic appearance of the vessels is the most important criterion of the differential diagnosis. In inflammatory lesions, the arterial channels remain orderly and taper in a normal fashion as they ramify in the tissues (Fig. 5, *A-C*). In contrast, malignant tumors



FIG. 1. Female, aged 50 years with pain and swelling of the left foot and ankle for 5 months. Clinical and roentgen findings (A) suggest either tumor or low grade infection, possibly tuberculosis. (B and C) Percutaneous transfemoral antegrade catheter arteriograms. B—arterial phase; C—venous phase. Findings typical of malignant tumor with (a) pathologic vessels, (b) tumor staining and (c) avascular areas of tumor necrosis. Diagnosis: Primary renal cell carcinoma.



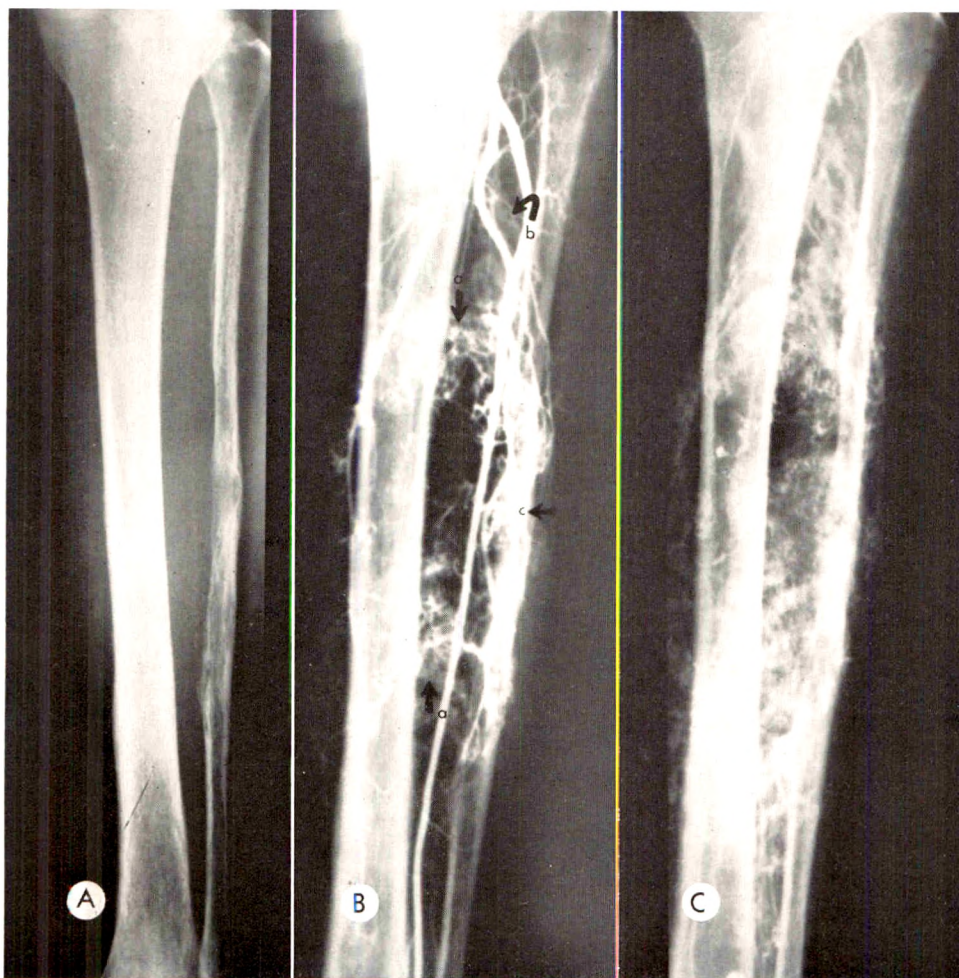


FIG. 2. Female, aged 70 years, with warm swollen leg for 4 months, clinically diagnosed as thrombophlebitis. (A) Bone destruction of the fibula and smooth cortical erosions of the tibia. (B and C) Percutaneous transfemoral antegrade catheter arteriograms. B—arterial phase; C—capillary venous phase. Highly vascular tumor with (a) pathologic vessels, (b) tumor staining and (c) abnormally early venous filling. Diagnosis: Malignant tumor, metastatic.

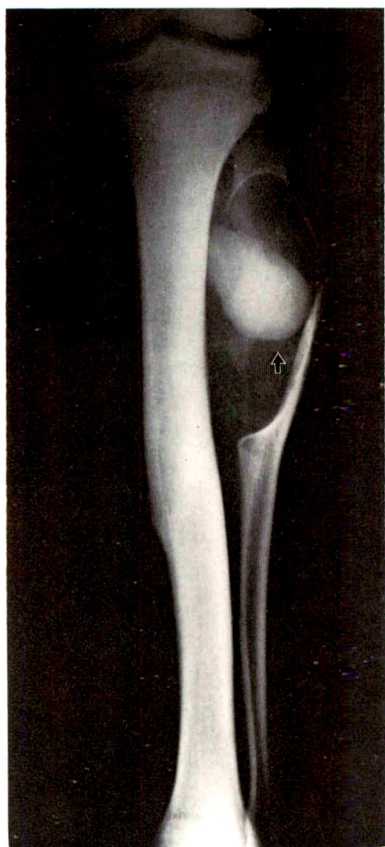


FIG. 3. Male, aged 16 years, developed acute pain in the left leg. Simple tibial fracture 3 years previously now securely healed. Femoral needle arteriogram shows large saccular aneurysm (arrow) of the anterior tibial artery. Diagnosis: Post-traumatic arterial aneurysm.

show pathologic vasculature of a variable degree, depending upon the lesion itself. (Fig. 1, A-C; 2, A-C; 7, A-C; 8, A-D; and 9, A and B). Pathologic vasculature is most vividly described by Strickland:¹⁴ "A pathologic vessel . . . is deployed seemingly without purpose, keeps no set course and shows no progressive diminution of caliber. As it runs through the mass of tumor its walls may be lined by tumor cells and in this instance, it ends its haphazard journey in amorphous spaces in the midst of areas of necrotic tissue. Such 'tumor lakes' will remain full of contrast medium for a considerable time there being no residual elasticity in their walls to aid the expression of the medium. . . ." The tumor vessel as described above appears quite specific for ma-

lignancy and we have not observed it in benign conditions. Staining, on the other hand, while very often a feature of vascular malignant tumors, may be seen in benign conditions. A classic example is the opacification of the vascular nidus of an osteoid osteoma as described by Lindbom *et al.*⁹

As is true of other specialized diagnostic procedures, the interpretation depends to some extent upon the previously determined clinical findings and upon the problem to be resolved. When arteriography is performed in a case of suspected osteoid osteoma, the purpose is to differentiate it from a bone abscess. Angiographic resolution of the problem lies in the demonstration of the avascularity of the abscess by persistence of a radiolucent area in the bone lesion, as contrasted with staining of the vascular nidus of the osteoid osteoma. In this instance staining within the bone lesion would not cause a misdiagnosis of malignancy. Additional support for the diagnosis of a benign lesion would come from interpretation of the character of the vessels supplying the lesion.

In our experience, angiography has been useful in the following conditions: (1) post-traumatic disruption and occlusion of major vessels, post-traumatic arteriovenous fistula (Fig. 4, A and B) and suspected post-traumatic arterial aneurysm (Fig. 3); (2) gigantism of an extremity for diagnosis of congenital arteriovenous malformation (Fig. 6, A, B and C); (3) bone infection, particularly low grade chronic osteomyelitis in which no clinical or laboratory clue to the diagnosis is present and a strong suspicion of bone tumor exists (Fig. 5, A, B and C); and (4) malignant bone tumors to corroborate the clinical impression of malignancy with angiographic evidence, and to determine the optimum site for biopsy of the lesion.

It is difficult to understand why arteriography has not been used to a greater extent in the investigation of trauma.¹ Clinical evaluation of patients has not infrequently resulted in exploration to determine whether or not the vascular system

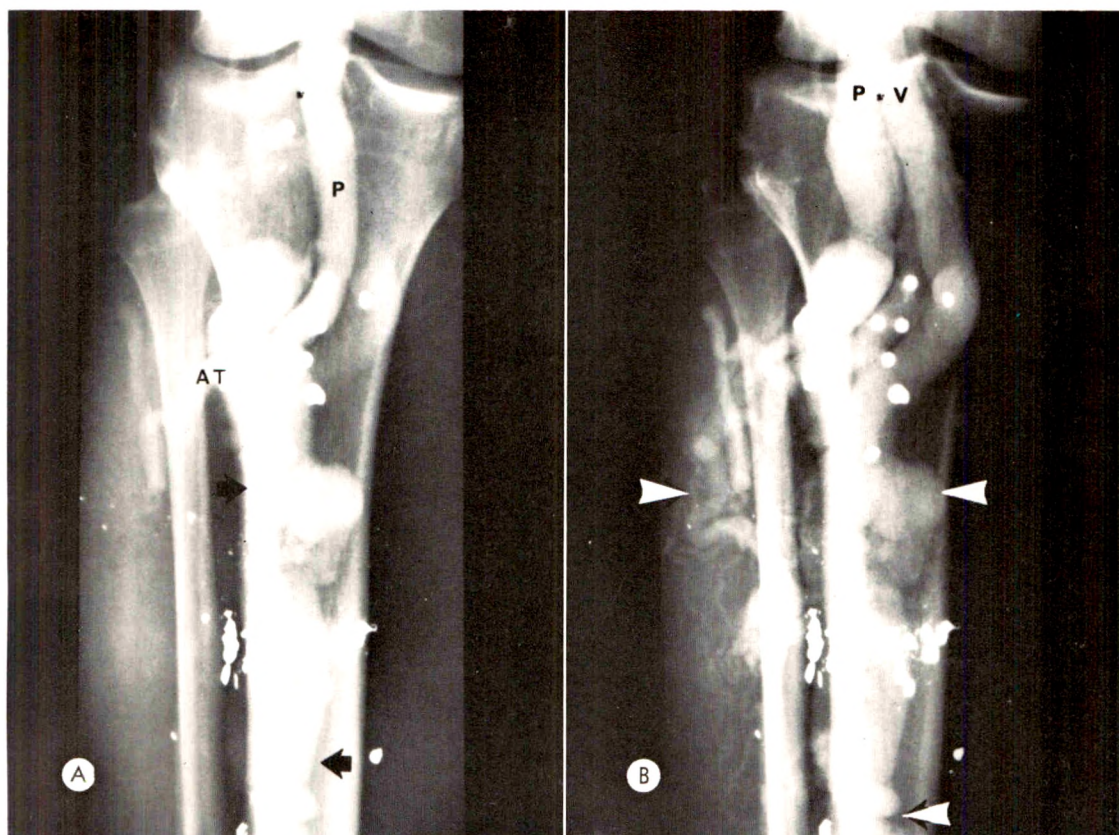
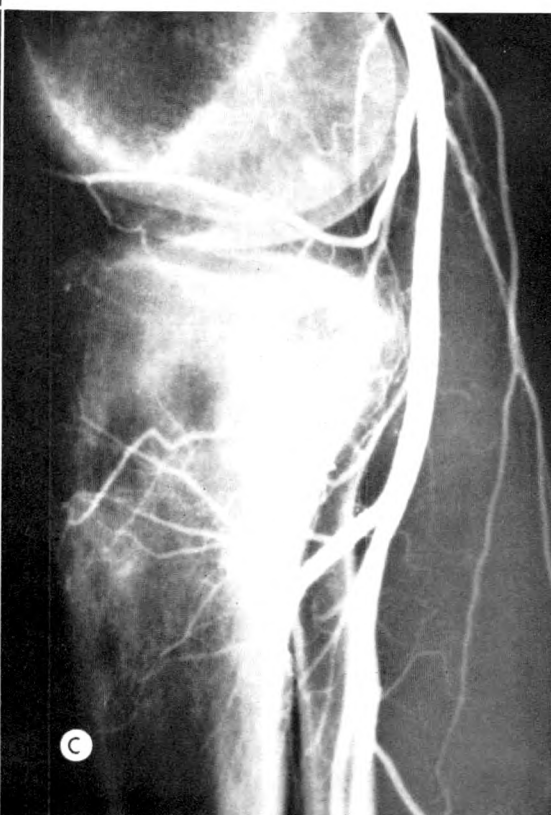
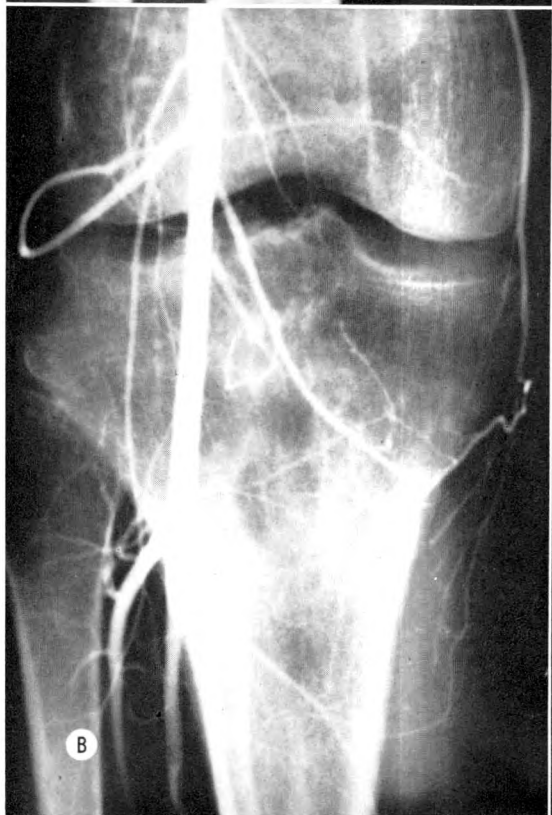


FIG. 4. Male, aged 59 years. Shot gun wound 42 years ago. Right leg somewhat enlarged, warm, discolored and pulsatile. No disability. (A) Femoral needle arteriogram shows enlarged popliteal (P) and anterior tibial (AT) arteries feeding a large complex arteriovenous fistula (black arrows). (B) Venous phase shows fistula (white arrows) and enlarged popliteal vein (P-V). Diagnosis: Post-traumatic arteriovenous fistula.



FIG. 5. Male, aged 57 years. Increasing pain in the right knee for 2 months with tender mass at the medial aspect of the proximal tibia. Normal laboratory studies. (A) Roentgenogram shows bone sclerosis in proximal tibia with areas of bone destruction. (B and C) Femoral needle arteriograms. B—anteroposterior; C—lateral. Normal vasculature. No evidence of malignancy (osteopoikilosis in distal femur). Diagnosis: Osteomyelitis, *Staphylococcus aureus*.



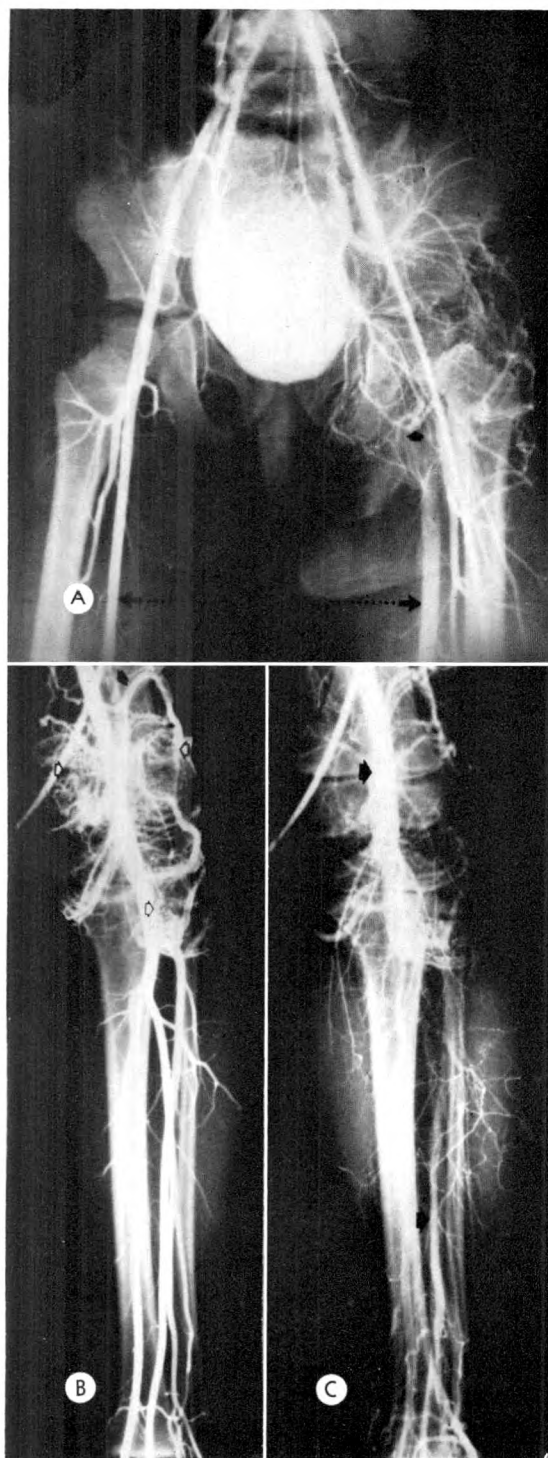


FIG. 6. Female, aged $2\frac{1}{2}$ years. Enlarged left lower extremity with increased warmth. (A, B and C) Percutaneous transfemoral catheter arteriograms. A and B—arterial phase; C—venous phase. The arterial bed of the hip and knee regions shows an

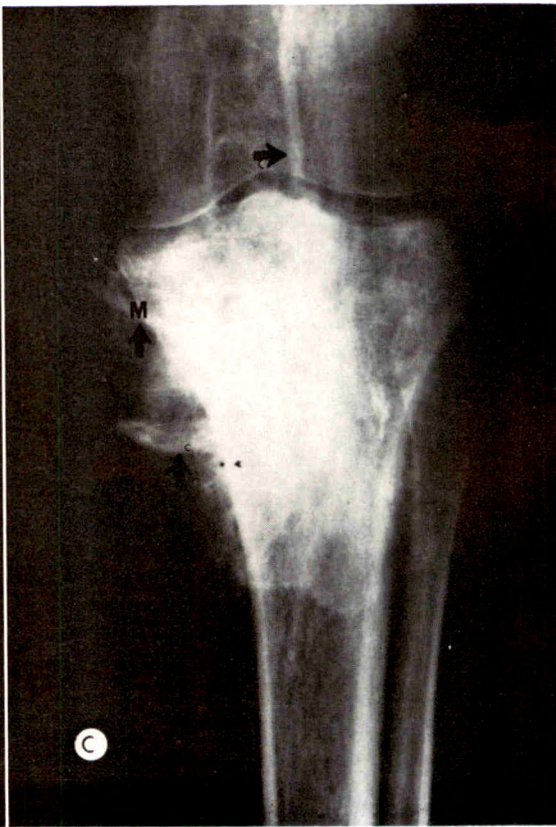
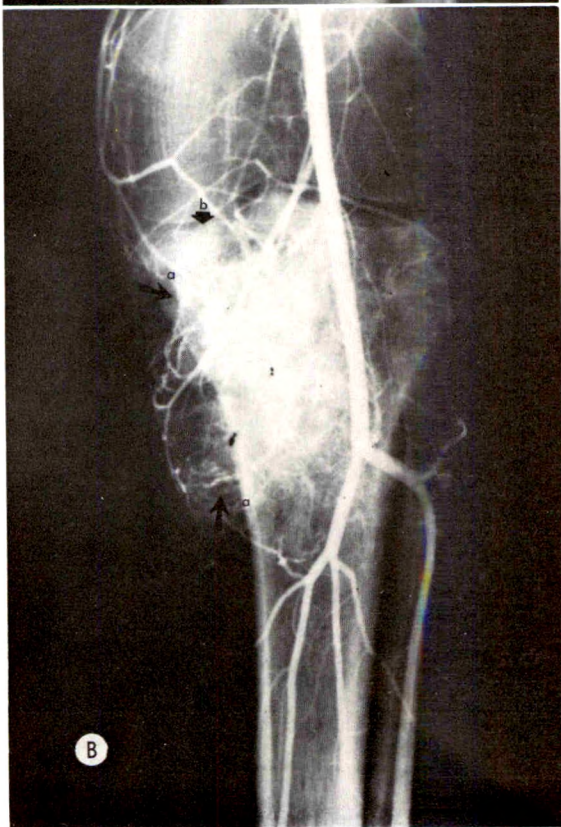
was intact and the extent of vascular injury, if present. This information can be safely and simply obtained through arteriography and normal studies of traumatized limbs will obviate exploration for this purpose. Detailed information as to the extent of vascular injury can be obtained, hematoma can be differentiated from arteriovenous fistulae and severance or occlusion of arterial channels can be demonstrated. There has been some investigation of the appearance of fractures which show a degree of increased vascularity of the tissues contiguous to a fracture site with normal progressive diminution in caliber of the vessels, characteristic of a benign condition.¹⁴ The vasculature distal to a fracture is normal. As evidenced by both traumatic aneurysm or arteriovenous fistula formation, the late results of trauma present a clear arteriographic picture.

Compression of the subclavian artery in the scalenus anticus syndrome can be confirmed by arteriography¹¹ (Fig. 10, A and B). Studies of patients presenting this symptom complex have been performed through a transfemoral approach by selective catheterization of one or both subclavian arteries. Arteriograms of the subclavian artery with the arm both adducted and abducted and externally rotated have shown narrowing of the lumen and alterations in the dynamics of flow. Patients with the clinical findings of the scalenus syndrome proven by arteriography have had excellent results from surgery. Along similar lines, there can be special instances in which arteriography is indicated. One patient demonstrated a critically placed osteochondroma which caused deviation and intermittent compression of the superficial femoral artery, resulting in claudication. Complete relief was obtained by resection of the osteochondroma.

abnormal increase in number and size of vessels with rapid shunting of blood into the venous system. Diagnosis: Congenital arteriovenous malformations.



FIG. 7. Male, aged 16 years. Left knee pain for 3 months. (A) Roentgenogram shows osteosarcoma of proximal tibia with bone destruction and exuberant tumor bone formation. (B and C) Femoral needle arteriograms. B—arterial phase; C—venous phase. Moderate number of tumor vessels (a) with tissue staining (b) and abnormally early venous filling (c) M—metaphyseal vein. Diagnosis: Osteogenic sarcoma.



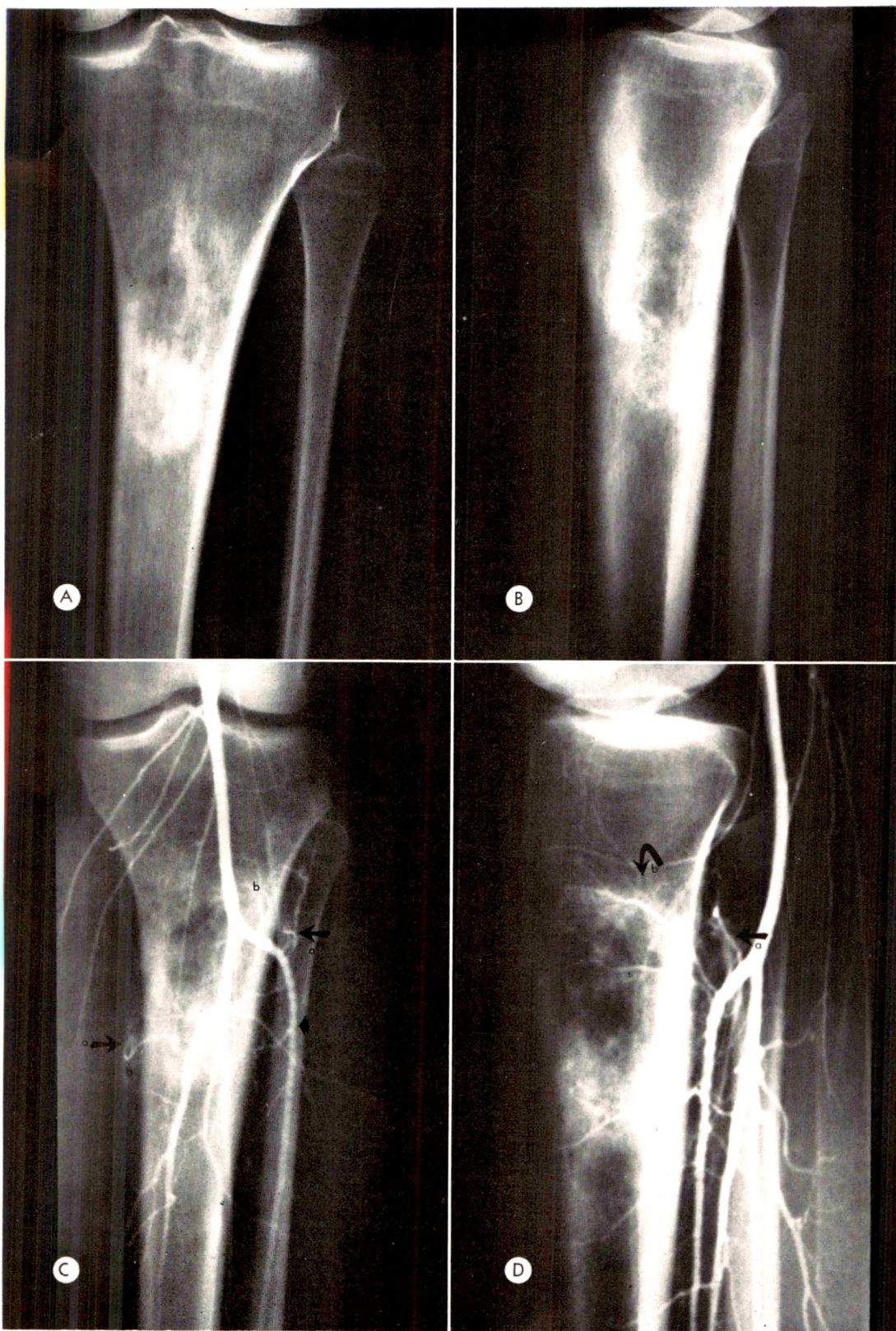


FIG. 8. Female, aged 32 years. Nontender mass noted at medial aspect of proximal tibia at time of pregnancy 2 months ago. (A and B) Roentgenograms show bone destruction and bone sclerosis with organized periosteal reaction. (C and D) Femoral needle arteriograms show sparsely vascular tumor with scattered tumor vessels (a) and tumor staining (b) seen in soft tissues and bone. Stationary waves present in anterior tibial artery (arrow). Diagnosis: Ewing's sarcoma.

Gigantism of a limb demands a detailed investigation as to etiology. Arteriography is indispensable in such circumstances, either to establish that there is no arteriovenous malformation, or when such a lesion exists, to provide visual detail of all abnormal arterial channels, and to demonstrate the location of the arteriovenous fistulae and the venous drainage from the affected part (Fig. 6, *A*, *B* and *C*). In our experience, surgery has not been beneficial in cases of extensive congenital arteriovenous malformations.

In discussing the need for correlation of routine roentgenographic and clinical information with an arteriogram, the means by which and importance of separating inflammatory processes from neoplasms have been mentioned. Apart from the mechanical displacement of vessels, we have not seen benign bone lesions, such as an enchondroma, with any significant vascular changes. The main features of the inflammatory process have been reviewed but additional mention should be made of the fact that the hypervascularity of inflammatory

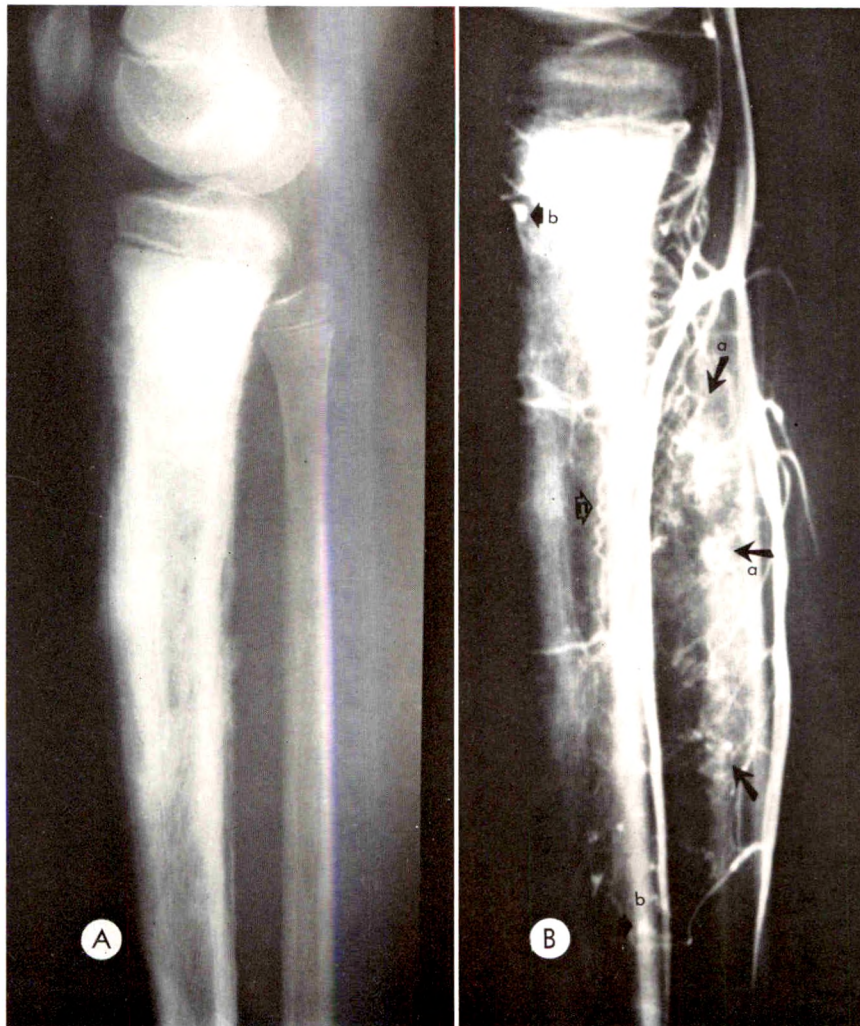


FIG. 9. Female, aged 9 years. Left leg pain for 3 months following minimal trauma and recent swelling of the leg. (*A*) Roentgenogram showing bone sclerosis and bone destruction with periosteal new bone formation. Pulmonary metastases were present. (*B*) Femoral needle arteriogram shows highly vascular malignant tumor with pathologic vessels (a) and vascular pools (b) with large soft tissue components. n—nutrient artery. Diagnosis: Ewing's sarcoma.

processes may result in earlier filling of the venous channels than is normal. This hemodynamic pattern, however, is not of the order of magnitude as that seen in malignant lesions. Angiographic observations in Paget's disease failed to reveal arteriographic abnormality with the exception of a mild increase in the vascular supply to the periosteum of the bone at a site contiguous to the active stage of the disease in the bone. The degeneration of Paget's disease and chondromatous or other benign tumors into their malignant counterparts is most often associated with clinical evidence of pain. Angiography in such conditions demonstrates the abnormal vasculature, thereby assisting in the determination of the ideal site for biopsy.

Lagergren *et al.*^{7,8} have made detailed investigations of the angiographic findings in osteosarcomas and fibrosarcomas. The clinical picture and physical findings of warmth and dilatation of subcutaneous veins in an area of an osteosarcoma are evidence of their high degree of vascularity. There seems to be some correlation between the degree of vascularity of such tumors, the extent of bone destruction, and the degree of malignancy of the tumor. More than half of the tumors studied showed that the soft tissue extension of the mass also had considerably increased vascularity. In only a small group was this pattern reflected in the medullary canal of the bone itself. Early shunting of contrast medium from arterial to venous channels is a feature of these vascular malignant lesions. In the fibrosarcoma group of tumors, some increase in vascularity was noted in the majority of cases while 25 per cent were essentially avascular. The degree of malignancy of the tumor appears to be directly related to the degree of vascularity. Many malignant tumors tend to be nonhomogeneous in their histologic structure and have varying degrees of growth rates. For this reason, choosing the site of biopsy of a tumor is of considerable importance. The arteriogram can be used to locate the most vascular portions of a tumor; these are apt to represent

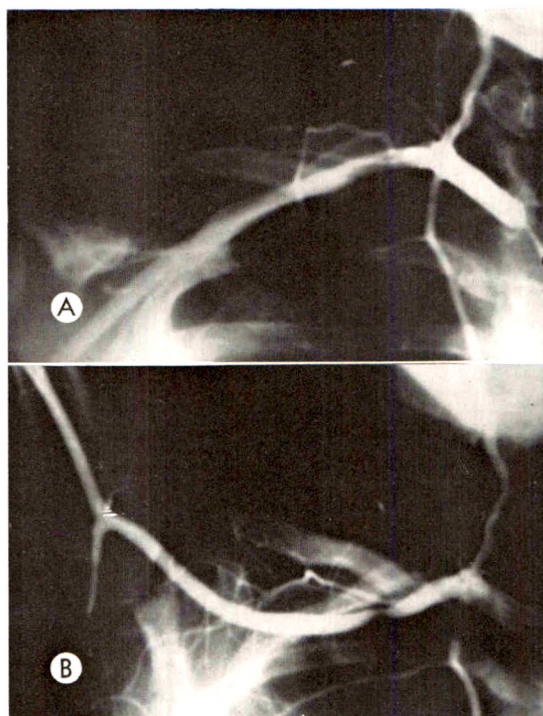


FIG. 10. Female, aged 10 years. Acute swelling, numbness, tingling and cyanosis of the right forearm and hand. (A and B) Percutaneous transfemoral catheter arteriograms. A—arm in adduction, (c) —catheter; B—arm in abduction. Segmental compression of the subclavian artery with the arm in external rotation and abduction. Scaleneotomy was performed with complete relief of symptoms.

the areas of the greatest rate of growth and the highest degree of malignancy.

Primary soft tissue sarcomas may show a profuse vascular network of pathologic vessels with early opacification of the venous drainage system and demarcation of the periphery of the tumor from the surrounding normal tissue. However, it may be difficult to precisely distinguish inflammatory from neoplastic processes, since some soft tissue malignant tumors are sparsely vascular. Cockshott and Evans,³ after an extensive study of soft tissue tumors, concluded that arteriography did not provide a great amount of additional information that was truly of value in enhancing diagnosis or in assisting in the therapeutic management of the patient. However, the vascular soft tissue lesions, such as hemangioma, glomus tumor and, of course,

the arteriovenous malformations fall into a different category; in these arteriography is indispensable. Metastatic lesions of both bone and soft tissue often present a vascular pattern identical to that seen in a primary lesion. There are no characteristics that permit the differential diagnosis between a primary and secondary lesion.

CONCLUSIONS

Arteriography is a simple and precise technique for the demonstration of vascular morphology in a variety of lesions seen in the practice of orthopedics. A broader application of this diagnostic procedure appears indicated, both for academic and practical reasons, to establish the role of angiography as an investigative method.

Arteriography can be of distinct value in the differential diagnosis of benign and malignant tumors, and can be of assistance in determining the proper site for a biopsy. In gigantism of extremities, it represents an indispensable diagnostic technique and in many other instances arteriography has proven to be of inestimable value in aiding diagnosis, treatment and prognosis.

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ASCENDING PHLEBOGRAPHY IN FRESH THROMBOSIS OF THE LOWER LIMB*

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THE appearance of post-thrombotic changes in the veins of the lower limb as seen at phlebography is well known. This cannot be said without reservation about the changes in fresh thrombosis. In 1940, Bauer² described 40 phlebographically examined cases of fresh thrombosis, all of which "showed a typically absent or defective opaque filling of one or all of the deep vessels draining the lower leg." Bauer also—as an exceptional finding—described the appearance of a floating thrombus in deep veins. However, he examined his cases with the leg horizontal. This technique does not permit a satisfactory contrast filling of the deep main venous trunks and the muscular veins of the calf. An "absent or defective" filling of one or all of the deep veins can be obtained also in patients with normal deep veins. A satisfactory study of the anatomy of these veins by phlebography can be made only with a vertical position of the extremity.^{5,7} There are few reports of phlebographic findings in cases of acute thrombosis made with the "vertical technique." Lindbom,⁸ who examined the patient in a sitting position, points out that in acute cases the thrombus frequently floats within the vessel and that the injected contrast material can be seen between the thrombus and the vessel wall. However, Lindbom⁸ does not present an account of any clinical material and it is not evident whether he shares the opinion of Bauer regarding the common phlebographic appearance of acute thrombosis.

Bauer selected for his examinations cases with clinical signs of acute thrombosis. This excluded cases which clinically run a silent course and it is improbable that cases of

very fresh, nascent thrombosis were included. In order to detect such cases, a group of patients in whom thrombosis is likely to occur has to be studied, irrespective of the presence or absence of clinical signs. Only one such series, by Hjelmstedt and Sundström,⁶ examined by phlebography with vertical positioning of the extremity has been published. These authors investigated the occurrence of thrombosis following fracture of the tibial shaft, but because of the plaster casts the muscle veins could not be studied. From autopsy data it appears that thrombosis usually starts in the calf and most likely in the muscle veins; however, a thrombosis confined to a muscle vein—as far as we have been able to ascertain—has not been demonstrated by phlebography.

A controlled clinical investigation of an anticoagulant drug (dicoumarol) with the use of phlebography provided the possibility of studying the thromboembolic disease in its early and silent stages. This trial was made on patients with a trochanteric fracture, which is the most common injury compelling elderly persons to remain recumbent. The clinical results of the trial have been published in an earlier paper. Here, mainly, those findings which are of roentgenologic interest are discussed.

MATERIAL

Originally, 58 cases with trochanteric fractures, 18 men and 40 women with a mean age of 73 years, were investigated. All patients were operated upon and the fractures were nailed. Four patients died before the phlebographic examination, which was usually made 3 weeks after the operation.

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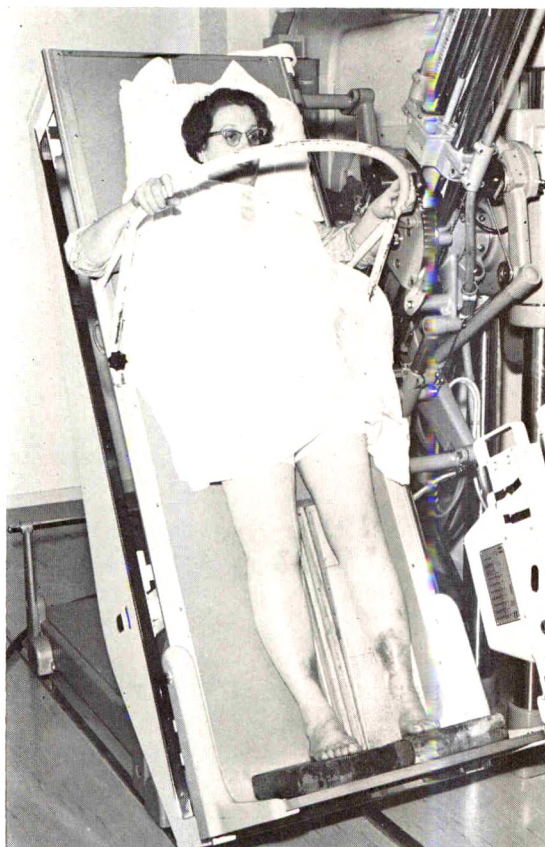


FIG. 1. Examination table with arm support and simple cassette holder.

TECHNIQUE

Phlebography was carried out in accordance with the technique described by Greitz.⁴ The patient was placed in the erect position, leaning backwards on an examination table which was tilted 60° degrees from the horizontal plane. A metal frame was attached to the examination table to help the patient maintain the erect position (Fig. 1). A vein of the dorsum of the foot was punctured percutaneously and 60 ml. of 45 per cent urografin was injected as rapidly as possible. Roentgenograms of the crus and thigh were obtained in two planes. Furthermore, studies of the upper thigh and pelvis were made in the frontal projection. This series was repeated so that at least 2 roentgenograms were made in each projection. No tourniquet or elastic bandage was applied. The roent-

genograms were exposed at 80 to 90 kv. In addition, intraosseous pelvic phlebography was carried out in the horizontal position following injection of 20 ml. of 45 per cent urografin into the greater trochanter.

RESULTS

Phlebography could be performed in only 44 out of the 58 patients. Four patients died before the examination. In 2 cases with serious mental disorders, phlebography was considered out of the question by a psychiatrist. In 8 cases attempts at phlebography were unsuccessful. In 3 cases the examination had to be interrupted or was unsatisfactory because of severe shock. Four patients with mental disorders did not cooperate sufficiently. In 1 patient it was impossible to puncture the veins of the foot.

In the 44 patients, good contrast filling was obtained of the deep and superficial veins of the calf, and of the popliteal, femoral and great saphenous veins. In 15 of these cases changes were seen on phlebograms which were considered due to acute thrombosis (Table 1). Thirteen of these patients, belonged to the untreated control group. (The significance of this finding as to the value of anticoagulant treatment is discussed in an earlier paper.) No pathologic changes were seen on pelvic phlebograms made in the horizontal position; all thrombotic changes were seen on phlebograms made with vertical positioning of the extremity. These changes consisted of filling defects located centrally in nonoccluded veins. The defects were all constant. Thus, they were visible and were of the same size and appearance on several roentgenograms, both in the lateral and the frontal projections (Fig. 2, A, B and C). They were all sharply outlined; as sharply as the vessel wall. Also, the proximal limits of the thrombi were quite sharp and often rounded. They formed casts of the veins in which they were located. These casts were even demonstrated in the venous valves (Fig. 2, A, B and C; and 3). As a rule, the defects were located centrally

TABLE I
LOCATION OF ACUTE THROMBOSIS ON PHLEBOGRAMS

Case No.	Muscular Veins of Calf	Peroneal Vein	Posterior Tibial Vein	Popliteal Vein	Femoral Vein	Deep Femoral Vein	Muscular Veins of Thigh	Superficial Veins
1	+	○	○	○	○	○	○	○
2	+	○	○	+	+	—	—	○
3	+	+	+	+	+	+	○	○
4	+	+	○	+	+	—	○	○
5	○	+	+	○	○	—	○	○
6	+	○	○	○	○	○	○	○
7	○	○	+	○	○	○	+	○
8	+	○	○	+	+	○	○	○
9	○	○	+	○	○	○	○	○
10	+	○	+	+	+	○	○	○
11	+	○	○	○	○	○	○	○
12	+	○	○	○	○	○	○	○
13	+	○	○	○	○	○	○	○
14	○	○	○	(+)	○	○	○	+
15	○	○	○	○	○	+	+	○

○ denotes vein filled with contrast medium, no thrombosis.

— denotes vein not filled.

+

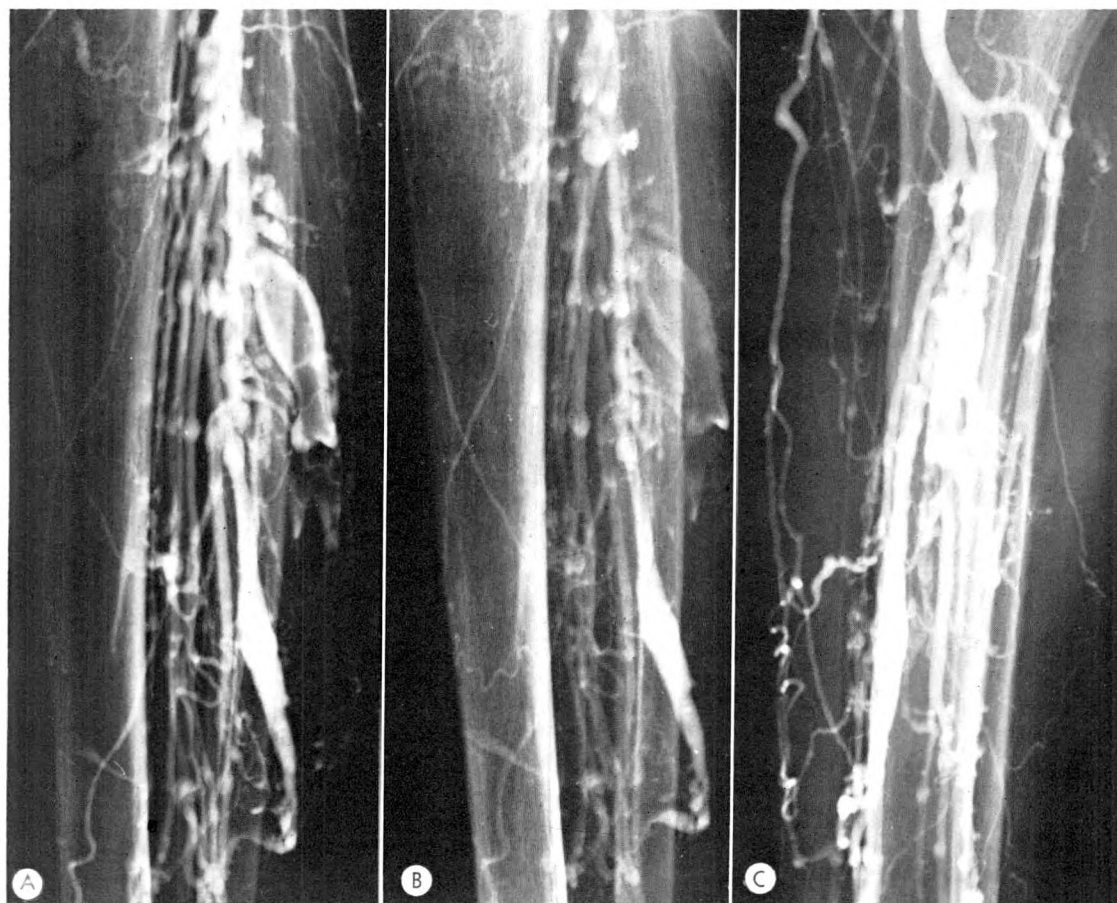
 denotes vein filled, thrombosis present.


FIG. 2. (A) Fresh thrombosis of muscular vein in the calf, forming a sharply outlined, centrally located filling defect. (B) Roentgenogram made about 1 minute later. The filling defect has a constant appearance, forming a cast of the vein. (C) The thrombus can be seen also in the anteroposterior projection.

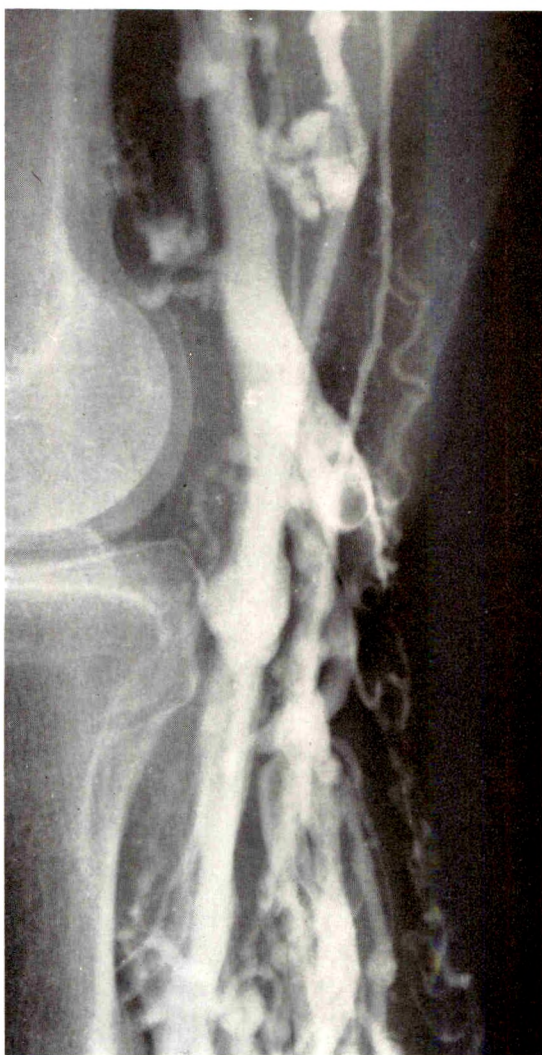


FIG. 3. Fresh thrombosis in proximal portion of small saphenous vein. The thrombus assumes the shape of the lumen of the vessel, including the venous valves.

within the vessel, but a somewhat eccentric position was also observed (Fig. 4, *A* and *B*). These findings make it possible to differentiate a thrombus from a defect caused by the inflow of blood, unmixed with contrast material, into a contrast filled vein. Such a defect is not constant, not sharply outlined, and usually not found in muscular veins but mostly in the superficial vein.¹

In the reported material the diagnosis of

thromboses in veins filled with contrast medium was always made on the observation of the characteristic filling defects and never on the basis of totally absent filling of venous channels.

In 10 of the 15 cases with positive findings at phlebography, the thrombosis was located in the muscular veins of the calf and in 6 of these there were no other signs of thrombosis. In 4 cases the deep veins of the calf were also involved and in 3 of these thrombosis was noted in the deep veins of the thigh. Of the 5 cases without thrombosis of the calf, the deep veins of the calf were involved in 3 cases (Fig. 5, *A* and *B*), the small saphenous vein in 1 case and the muscular veins of the thigh, including the distal portion of the deep femoral vein, in 1 case. The muscular veins in the thigh were thrombosed in 2 cases. Thus, muscular vein thrombosis was found in 12 out of the 15 cases. Five pa-

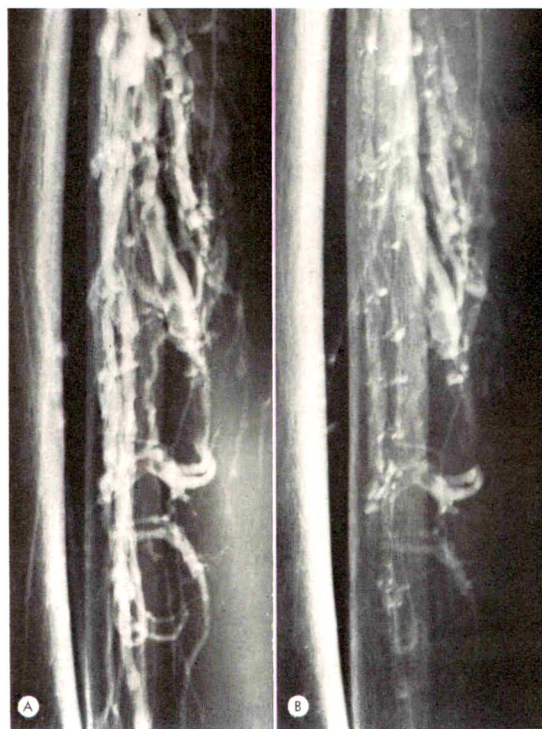


FIG. 4. (*A* and *B*) Thrombus in muscular vein is somewhat eccentrically located. The defect has a constant appearance.

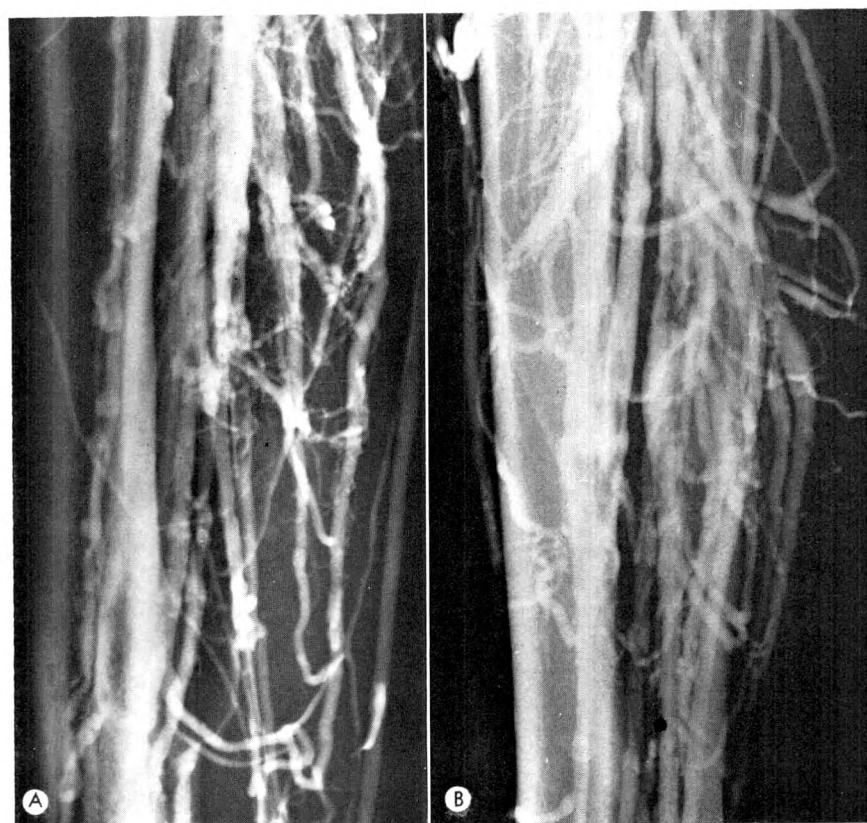


FIG. 5. (A and B) Small constant filling defect of posterior tibial vein, diagnostic of thrombosis, is seen at the same level in both the lateral and frontal projection.

tients, 1 with and 4 without acute thrombosis, showed signs of old recanalized thrombosis of the deep veins.⁵

DISCUSSION

As the phlebographic diagnosis of acute thrombosis was not correlated to the results of any other method, the accuracy of the phlebographic method can be questioned. Only in 1 patient was the roentgenologic diagnosis confirmed at autopsy. However, the clinical investigation in which these patients participated supplied indirect evidence for the reliability of phlebography. No information regarding clinical signs of thrombosis was given to the radiologist, who referred all cases of thrombosis but 1 to the non-dicoumarol treated group. With an unreliable method, the roentgenologist would not have been

able to detect a difference between 2 groups of cases unknown to him. It would have not been possible by using an unreliable method to classify all cases of thrombosis into one group.

All patients in this series were examined with regard to clinical signs (Table II). There were 10 cases with clinical signs of thrombosis; phlebography could be performed in 8. Of these, 7 showed thrombosis on phlebograms. On the other hand, out of 12 surviving patients with phlebographically proven thrombosis, only 6 had clinical signs. This provides the previously missing evidence that thrombosis often runs a silent course, not only—as autopsy studies have shown—among those who succumbed to the diseases but also among the surviving patients. Finally, we wish to point out that the frequency of thrombo-

TABLE II
CLINICAL AND PHLEBOGRAPHIC DIAGNOSIS
OF THROMBOSIS

		Phlebographic Diagnosis			
		+	—	miss- ing	
Clinical	+	7*	1	2	10
Diagnosis	—	8	28	12	48
		15	29		

+ denotes thrombosis present; — denotes thrombosis absent.

* One patient died later; thrombosis was found at necropsy.

sis in this series as demonstrated by phlebography, high as it may seem, is presumably still higher, since only one leg was examined.

CONCLUSIONS

1. Early thrombosis can be detected by ascending phlebography with the leg in a vertical position. The method is sufficiently reliable to be used in clinical investigations in which the frequency of the thromboembolic disease is to be studied.

2. In fresh thrombosis a floating thrombus is usually demonstrated on phlebograms.

3. Muscular vein thrombosis can be shown on phlebograms of a vertically placed extremity and is a frequent finding in cases with fresh thrombosis of the lower leg.

4. Phlebographic studies provide the previously missing evidence that thrombo-

sis of the lower extremity frequently runs a silent course also in surviving patients.

SUMMARY

Acute thrombosis of the lower limb has been studied with ascending phlebography in the erect position. The thrombosis is frequently confined only to muscular veins. Its roentgenologic features are described.

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SELECTIVE VASODILATATION AS AN AID TO ANGIOGRAPHY*

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THE slow blood flow through many of the smaller arteries has imposed more critical limitations on successful arteriographic visualization than their size. Vessels remain unseen on good serial studies, when they could be readily filled with contrast material, if they were carrying their maximum amount of blood. Since various vasodilator drugs and mechanisms are available for temporarily increasing the blood flow in selected areas, we have investigated the clinical usefulness of such methods for improving the diagnostic quality of our arteriograms. We have been interested not only in the peripheral circulation, but in visceral branches such as the celiac and bronchial arteries. We have used tolazoline, histamine, papaverine, lidocaine, and the reactive hyperemia following arterial occlusion.

MATERIALS AND METHODS

Following routine serial arteriographic studies by the percutaneous catheter method, under local anesthesia a slow intra-arterial injection was made of one or more of the following: tolazoline hydrochloride, 10–50 mg.;* papaverine hydrochloride, 30–60 mg.;† lidocaine hydrochloride, 60–100 mg.;‡ and histamine acid phosphate, 0.1–0.2 mg.§ Each of the medications was diluted to 15–30 cc. with normal saline. Alternatively, a blood pressure cuff was inflated on a previously elevated limb to above systolic pressure for 7 minutes, ac-

* Priscoline hydrochloride, Ciba Pharmaceutical Co., Summit, N. J.

† Papaverine hydrochloride, Wm. S. Merrill Co., Cincinnati, Ohio.

‡ Xylocaine hydrochloride, Astra Pharmaceutical Products, Inc., Worcester, Mass.

§ Injection histamine acid phosphate, Eli Lilly & Co., Indianapolis, Ind.

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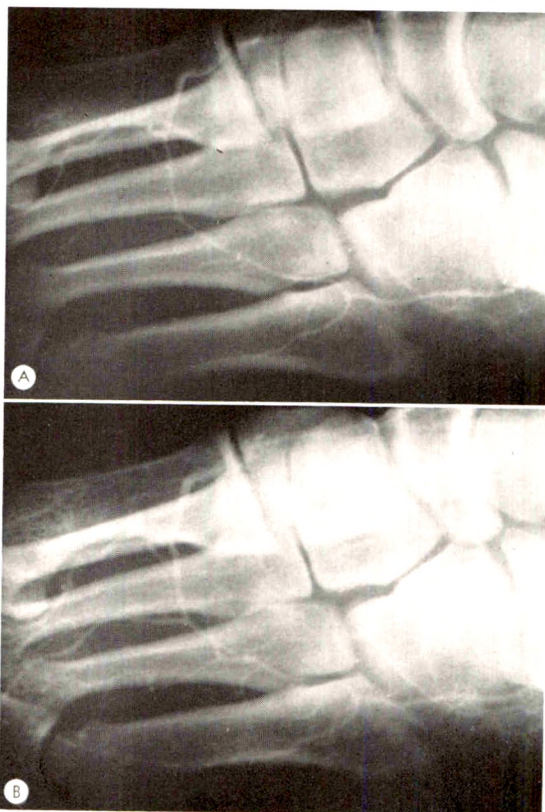


FIG. 1. Thirty-seven year old male with leg edema. Left femoral arteriogram showed no evidence of arterial disease. (A) Maximal visualization of arteries to the foot, 13 seconds after injection of 15 cc. methylglucamine diatrizoate 60 per cent in left external iliac artery. Only a few of the metatarsal arteries are seen. (B) Similar contrast injection after introduction of 25 mg. tolazoline. Maximal visualization 4 seconds after injection. The metatarsal arteries are well filled and dilated.

cording to the method of Hishida.¹⁰ Serial roentgenograms were then made to duplicate as closely as possible a series made before vasodilatation. The femoral artery was studied in 35 patients, the brachial in 7, the celiac in 7, and the bronchial in 3

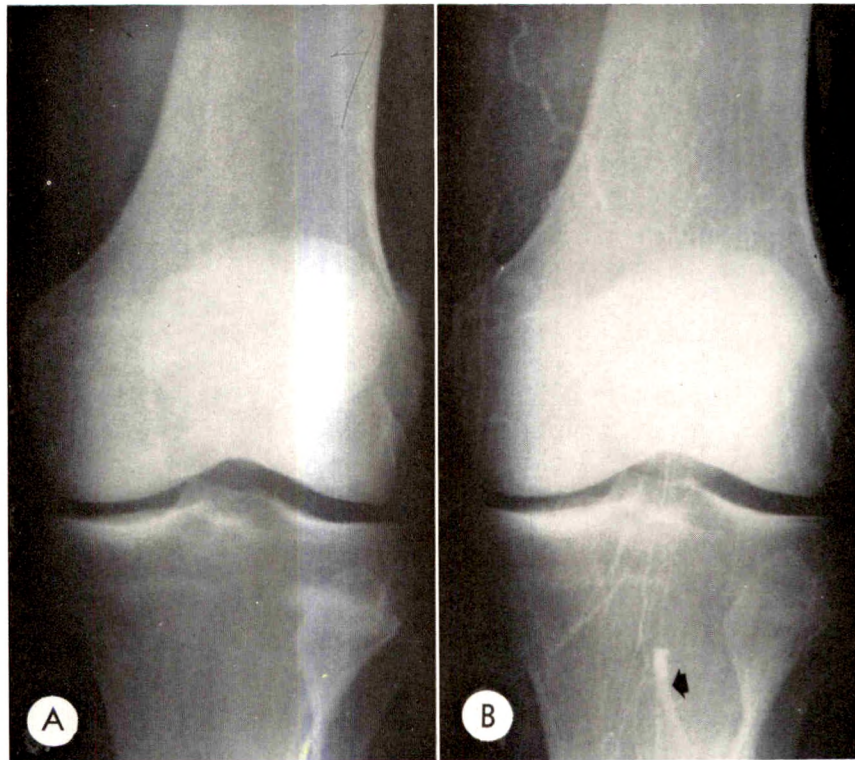


FIG. 2. Fifty-nine year old male with claudication in the left leg. Extensive bilateral iliac arteriosclerosis and complete occlusion of the left superficial femoral artery at its origin were demonstrated. Serial roentgenograms (A) from 6 to 26 seconds after injection of 30 cc. methylglucamine diatrizoate 76 per cent in the distal aorta show no filling of vessels to the left leg. (B) Similar contrast injection after introduction of 20 mg. tolazoline in left external iliac artery shows collateral vessels reconstituting a short segment of the distal popliteal artery (arrow), 13 seconds after injection.

cases. Blood pressure and pulse were checked frequently during the procedure.

RESULTS

In the lower extremity, we noted no significant change following intra-arterial administration of papaverine or lidocaine. The effect of tolazoline seemed to vary with the condition of the arteries. In those patients with relatively good circulation to the foot, there was a two or three fold increase in the speed of flow and much better visualization of the distal small vessels (Fig. 1, *A* and *B*). In patients with poor circulation to the foot, but with extensive collateral vessels at the level of the thigh and knee, these vessels became markedly dilated, and the runoff arteries in the leg could be more clearly delineated (Fig. 2, *A* and *B*). In patients with femoral artery obstruction and poor collateral circulation, tolazoline produced little effect. Injection during the phase of reactive hyperemia produced visualization of the deep arteries of the leg even in some patients with markedly impaired circulation (Fig. 3, *A* and *B*), but was completely ineffective in others.

In the upper extremity we noted a dramatic increase in blood flow to the hand both following tolazoline injection, (Fig. 4, *A* and *B*) and release of arterial compression. The latter method proved particularly useful in a patient in whom the digital circulation had to be studied via an injection in the proximal subclavian artery (Fig. 5, *A* and *B*).

In the celiac axis both tolazoline and histamine produced increased flow through the splenic artery (Fig. 6, *A* and *B*), and improvement of the portal phase of the angiogram (Fig. 7, *A* and *B*). Histamine appeared to be slightly superior. The hepatic artery was also dilated in some cases, but paradoxical constriction was present in others. In the bronchial artery the injection of tolazoline produced in all cases an increase in the size and filling of branch vessels (Fig. 8, *A* and *B*).

Some of the patients complained of chills after tolazoline and a characteristic local

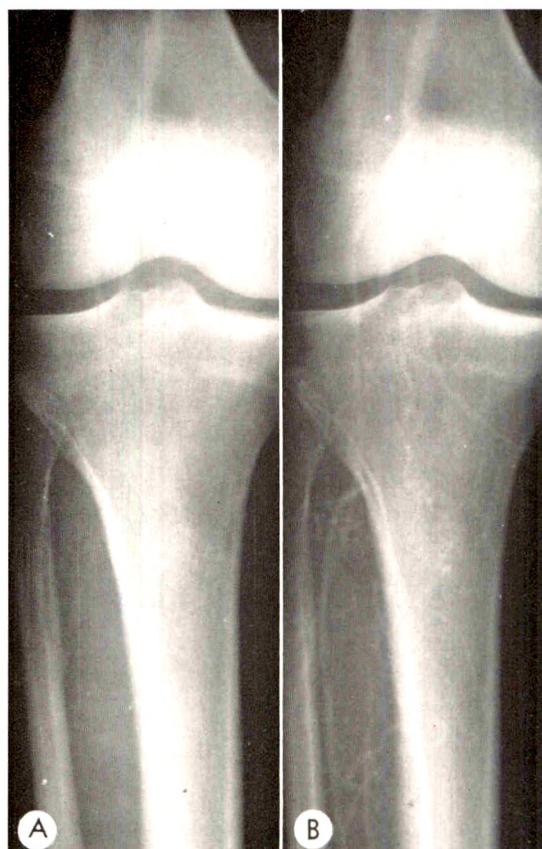


FIG. 3. Fifty-two year old male with absent femoral pulses bilaterally. Translumbar aortogram showed complete occlusion of the right external iliac and left common femoral arteries. (*A*) Maximum visualization of arteries to right leg 20 seconds after pressure injection of 30 cc. methylglucamine diatrizoate 76 per cent in mid-abdominal aorta. Very little runoff distal to the occluded popliteal artery is seen. (*B*) Similar injection immediately after release of blood pressure cuff just below knee, inflated to above arterial pressure for 7 minutes. Runoff vessels to the leg, including an intact anterior tibial artery, are well seen 7 seconds after injection.

reddening of the skin was observed following peripheral injection. The slight hypotensive effect was well tolerated by all. Papaverine, histamine, lidocaine and tourniquet ischemia produced no side effects. There were no complications.

DISCUSSION

The principal peripheral vasodilator drugs can be grouped into 4 classes.¹ Tolazoline is in the category of agents which act by com-

petitive blocking of vasoconstrictor (epinephrine) receptors in the arterial wall. Papaverine is representative of the myovascular relaxants, which have a direct paralyzing action on the smooth muscle of blood vessels. While histamine is listed in the same category, its action, particularly in relation to physiologic vasodilator mechanisms, is still uncertain.¹⁸ Lidocaine acts both as a myovascular relaxant and, in the third group, as a vasomotor center depressant. The fourth category consists of ganglion blocking agents such as hexamethonium bromide. The vasodilating effect of ischemia is possibly a still different mechanism, related to the release by the tissues of a polypeptide similar to bradykinin.^{3,8,9}

The therapeutic intra-arterial administration of vasodilator drugs has been well documented.^{4,14} Their usefulness is limited by their short period of action, their tend-

ency to dilate arteries to the skin rather than to the muscles, and the difficulties of administration.

The use of intra-arterial vasodilators for arteriography was first reported in 1937 by Sgalitzer¹⁹ who used papaverine, apparently without much success. Both he and Ratschow¹⁶ found the reactive hyperemia following arterial compression useful. The latter author also used diathermy and sympathetic block with some benefit. In recent years, although there was considerable interest in coronary^{7,11,12} and cerebral vasodilators,^{5,6} little attention was paid to drugs acting on the remaining visceral and peripheral vessels. Nordenström,¹⁵ in his Chamberlain lecture on methods of altering circulatory dynamics to improve roentgen examination of the cardiovascular system, disregarded the subject almost entirely except for a brief mention of sodium acetri-

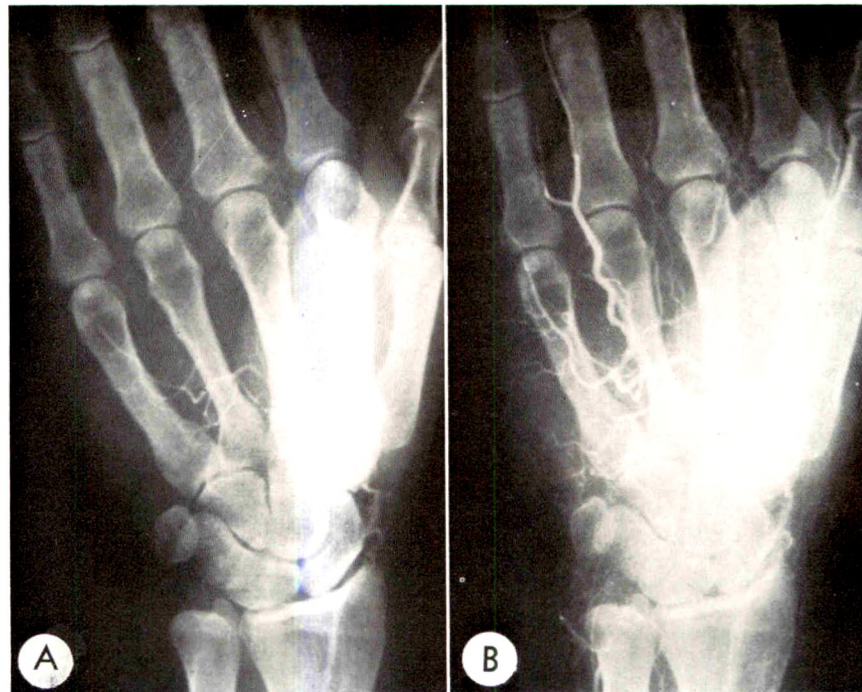


FIG. 4. Fifty year old female with recent onset of Raynaud's phenomenon and diminished wrist pulses. (A) Maximum visualization of arteries to left hand 21 seconds after injection of 10 cc. lidocaine hydrochloride 1 per cent and 15 cc. methylglucamine diatrizoate 60 per cent in left brachial artery. Only a few small metacarpal branches are seen. (B) Similar injection after introduction of 20 mg. tolazoline in left brachial artery. The irregular cutoff of metacarpal and digital arteries and collateral network in the wrist and palm are now demonstrated, 6 seconds after injection.

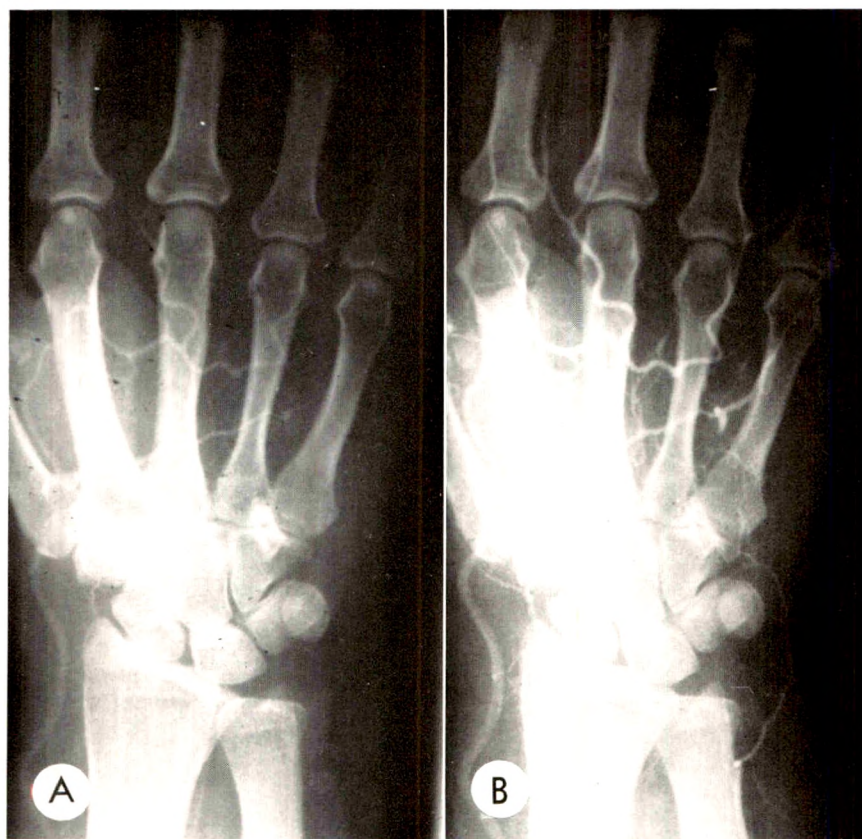


FIG. 5. Fifty-one year old female with recurrent nonhealing infections of the fingers and markedly diminished wrist pulses. The brachial and axillary pulses could not be felt because of the patient's obesity. Only the proximal portion of the left subclavian artery could be catheterized via the retrograde femoral route. The axillary and brachial arteries were small but no occlusion of the proximal vessels was seen. (A) Maximum visualization of the arteries of the left wrist and hand, 14 seconds after injection of 15 cc. methylglucamine diatrizoate 60 per cent in left subclavian artery. The ulnar artery and digital branches are not seen. (B) Similar injection immediately after 7 minutes of arterial occlusion by sphygmomanometer cuff. The occlusion of the distal ulnar artery and the patchy absence of digital arteries are seen, 3 seconds after injection.

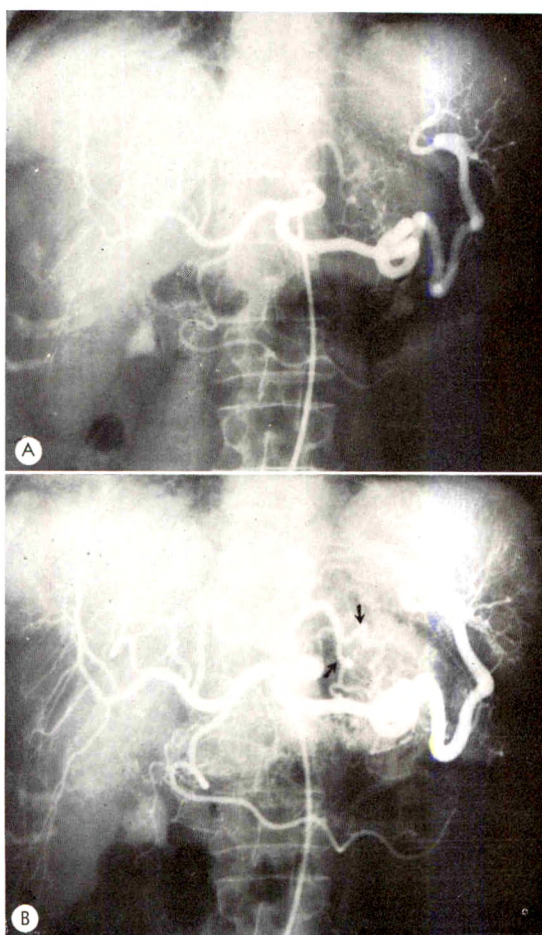


FIG. 6. Sixty-eight year old female with advanced gastric carcinoma. (A) Celiac arteriogram with 40 cc. methylglucamine diatrizoate 76 per cent gives fair visualization of the hepatic and splenic vessels. (B) Similar injection after 0.15 mg. histamine acid phosphate shows dilatation of all branches. The smaller hepatic arteries are well filled. The spleen is enlarged in comparison to the first injection. Branches of the left gastric artery supplying the lesser curvature tumor can be clearly seen (arrows).

zoate as a vasodilator. Hishida,¹⁰ in an extensive series, found tolazoline much less effective than his method of reactive hyperemia; however, there is some doubt whether he injected the drug sufficiently slowly to produce maximum local action.

In evaluating the effect of vasodilators in arteriography, many variables must be considered. The contrast media used for angiography are themselves vasodilators.¹⁷

However, methylglucamine diatrizoate 60 per cent and 76 per cent,* which we used, act much more weakly and for a shorter period than sodium acetrizoate¹³ and had no residual effect in control experiments with injections at 10 minute intervals. General anesthetics produce profound changes in vascular tone² which can be demonstrated angiographically.²⁰ We limited ourselves to local anesthesia and light sedation. Such factors as changes in room temperature, blood pressure and the presence of spasm in the larger vessels due to catheter manipulation must also be evaluated.

The time of injection of the vasodilator

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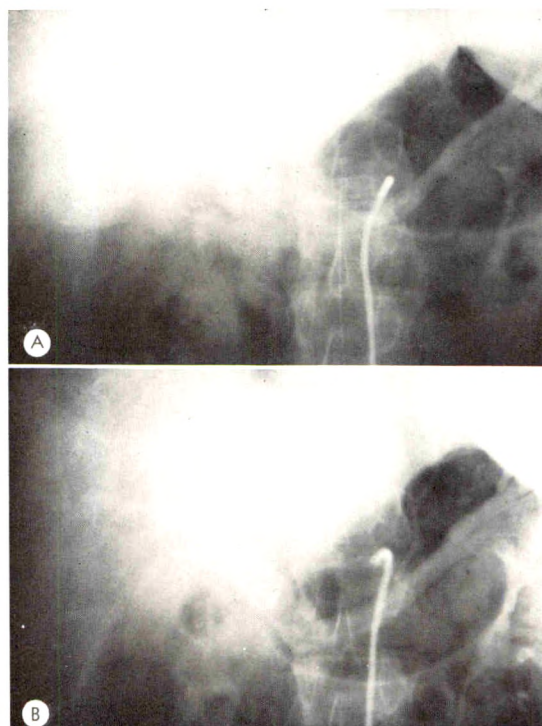


FIG. 7. Fifty-eight year old male with bronchogenic carcinoma being evaluated for hepatic metastases. The arterial phase of the celiac arteriography was normal. (A) Best visualization of the portal system 15 seconds after injection of 35 cc. methylglucamine diatrizoate 76 per cent in celiac axis. (B) Similar injection after introduction of 0.18 mg. histamine acid phosphate in celiac artery. The splenic and portal veins are well seen, 10 seconds after the injection. Note the dilatation of these vessels compared to the first study.

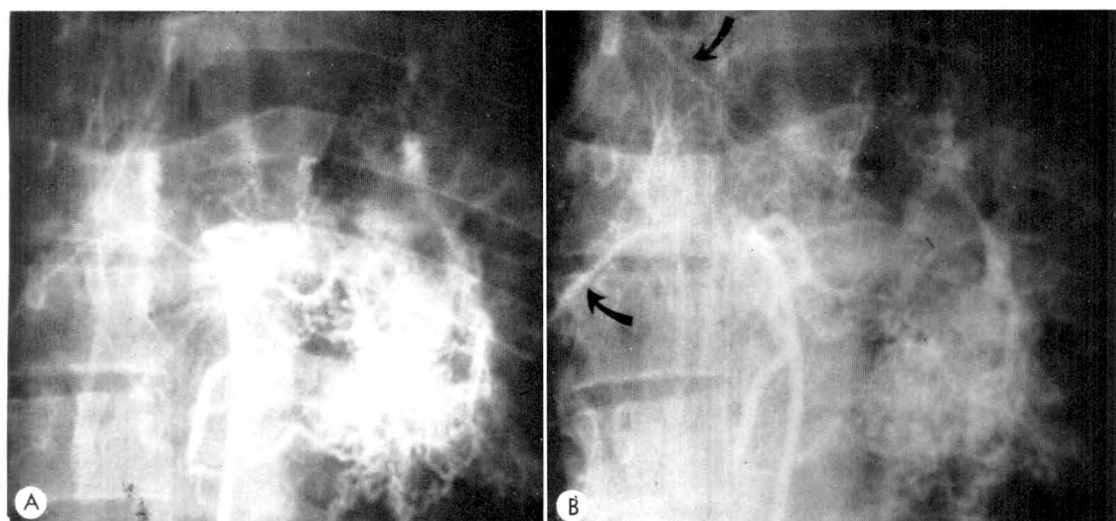


FIG. 8. Fifty-five year old male with bronchogenic carcinoma (A) Selective arteriogram of the left lower bronchial artery with 10 cc. methylglucamine diatrizoate 60 per cent shows dense tumor stain. Only a few filamentous normal branches are seen. (B) Similar injection after introduction of 10 mg. tolazoline into bronchial artery. A left upper bronchial artery is now filled (upper arrow), and the right bronchial branch (lower arrow) is dilated. The better filling of normal branches diminishes the contrast of the tumor stain.

is apparently quite critical and administration in less than 5 minutes results in more systemic and less local effect. We found that arteriography within several minutes is sufficiently fast to demonstrate the vasodilatation, but that by the time 15 minutes have elapsed there is some decrease of effect of tolazoline. We are planning to study dose and time factors more completely in the laboratory.

The use of vasodilators can obviously enhance the angiographic study of peripheral vessels. Visualization of visceral arteries with vasodilators is a greater challenge. Celiac arteriography can reveal some abdominal tumors, some sites of bleeding, some esophageal varices and some hepatic metastases. The yield of success may be considerably increased by the appropriate application of vasodilators. Superior and inferior mesenteric arteriography may also be improved by the same method. Bronchial arteriography has recently become feasible and the dilatation of these vessels during angiography may afford the opportunity to delineate peripheral lesions. We feel that there is a real need in the expand-

ing field of selective angiography to utilize fully all available pharmacologic methods to improve visualization of blood vessels.

SUMMARY

Selective intra-arterial injection of several vasodilator drugs and vasodilatation following ischemia have enhanced visualization of small arteries in peripheral, celiac and bronchial arteriography.

In 35 lower and 7 upper extremity arteriographies, tolazoline and reactive hyperemia proved helpful in improving visualization of peripheral vessels, while papaverine and lidocaine were not effective. In 7 celiac arteriographies, histamine and tolazoline increased splenic artery filling and improved the portal venogram. In 3 bronchial arteriographies, tolazoline dilated the normally small bronchial arteries. The various clinical applications of these techniques are now being investigated.

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A ROENTGENOLOGIC APPROACH TO THE MEASUREMENT OF VENOUS BLOOD FLOW

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THE measurement of the blood flowing through vital organs could furnish important clinical information. Unfortunately, the methods commonly employed to measure blood flow are either not accurate enough or are too complex for easy clinical utilization. The simple approach described below was found accurate in vascular models, was used in dogs and, with the proper instrumentation, could be applied to humans. The method is based on hemodynamic effects which can be visualized by the roentgen rays.

PROCEDURE

A catheter is introduced counter flow in the chosen vein. This catheter has two unequal channels. The smaller channel serves to inject a radiopaque medium slowly and continuously. This smaller channel has a side opening 1 cm. from the tip. The larger channel serves to withdraw blood at a gradually increasing rate by means of a manually operated syringe. The catheter is large enough to allow the withdrawal of blood at the maximum expected rate without vacuum formation and small enough not to obstruct the vein. Figures 1 and 2 show such a catheter in the inferior vena cava of a dog.

DISCUSSION

If no blood is withdrawn through the larger channel, the radiopaque medium, which is injected continuously, is carried away by the blood flowing between the inner end of the catheter and the walls of the vein (Fig. 1). When blood is withdrawn through the large channel at a gradually increasing rate, the flow of blood between the inner end of the catheter and the walls of the vein becomes slower and slower. When

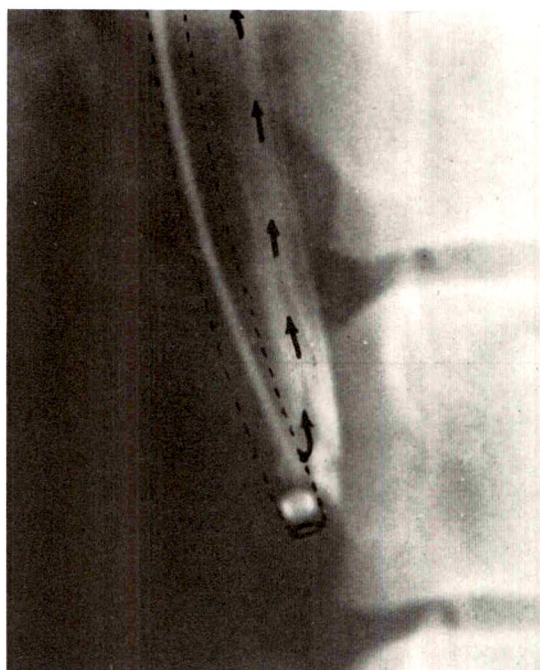


FIG. 1. Radiopaque material flushed toward the heart when no blood is withdrawn through the large channel.

all the blood coming from the periphery is sucked into the large channel, the blood between the inner end of the catheter and the venous walls ceases to flow altogether. While this blood slows down and finally stops, the contrast medium which is continuously discharged into it opacifies it to a maximum density. A long opaque bolus becomes visible at fluoroscopy (Fig. 2). At the moment this bolus is stationary, and it can be kept stationary by stabilizing the rate of withdrawal. This is not difficult to do under fluoroscopy because if the rate of withdrawal becomes excessive the bolus moves slowly backwards and is sucked into the catheter, while if the rate is insufficient the bolus moves slowly toward the heart.

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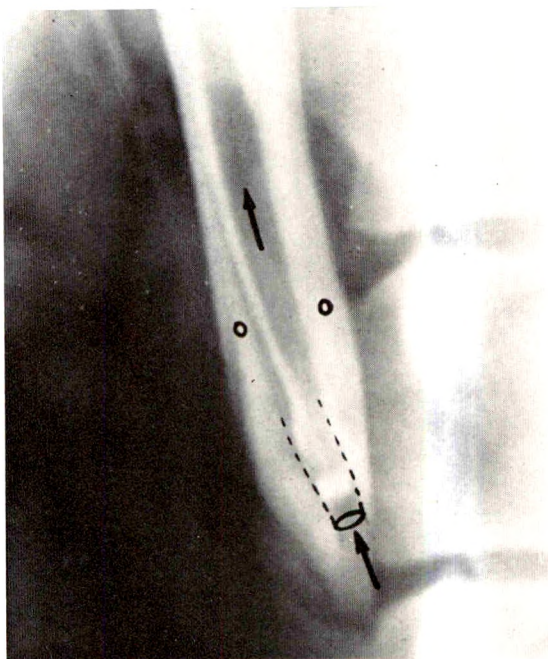


FIG. 2. Stationary opacified blood surrounding the end of the catheter when all the peripheral blood is withdrawn through the large channel.

A stationary bolus occurs only when one withdraws exactly the amount of blood which would have normally flowed through the vein. The venous blood flow can thus be measured at the extracorporeal end of the catheter.

CONCLUSION

The method is independent from the diameter of the vein and from the speed of blood flow and no errors are introduced by the catheter or by measuring instruments. With proper sterility, the blood withdrawn can be reinjected so that no blood loss is involved.

Electronic instruments, now available, should make it possible to employ this method in humans with a minimum of manipulation and radiation exposure.

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SEGMENTAL OCCLUSION OF THE MIDDLE CEREBRAL ARTERY

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ARTERIOGRAPHIC studies of patients with the stroke syndrome have become commonplace. However, most of the emphasis has been placed upon occlusion of the extracranial portions of the carotid and vertebral arteries, with relatively little attention given to intracranial obstruction of these vessels and their branches. Occlusions of the middle cerebral artery, either complete or limited to one of its major segments, can be identified without difficulty on carotid arteriograms. Publications concerned with segmental occlusion of the middle cerebral artery, however, have appeared infrequently. One of the largest series to date is that presented by Jacobsen and Skinhøj.² The diagnosis of total or segmental occlusion was made by arteriography in 36 of their patients. The obstruction was complete in 14 cases, limited to the ascending frontal artery in 10, and to the sylvian arteries in 12.

The complexity and variability of the middle cerebral branches make the positive identification of segmental occlusion difficult. The pertinent anatomic relations and variations have been discussed in other publications and will not be reviewed in detail here.^{1,3,4} For roentgenologic purposes, the middle cerebral artery has 4 major named branches distal to the origin of the lenticulo-striate arteries: (1) the ascending frontal, (2) the posterior temporal, (3) the posterior parietal, and (4) the angular arteries. The ascending frontal artery is readily identified in the lateral projection since it occupies a relatively clear space between the pericallosal artery above and the sylvian vessels below (Fig. 1A). The anteroposterior projection is less helpful, although frequently confirmatory

evidence is obtained from these films (Fig. 1B). Occlusion of the sylvian arteries can easily be recognized on the lateral projection by the characteristic absence of arteries in the temporal and inferior parietal areas (Fig. 2A). Roentgenograms in the anteroposterior projection also provide information of diagnostic value, since with occlusion of the sylvian arteries a characteristic avascular zone is present in the inferior temporal region (Fig. 2B). On occasion, this may be sufficiently marked to mimic the appearance of a space occupying lesion inferiorly situated in the middle cranial fossa. The other major named middle cerebral branches are frequently difficult to distinguish with assurance and overlap considerably. We have, therefore, not attempted to distinguish occlusion of the individual arteries distal to the origin of the ascending frontal artery, but have considered these as a unit which we shall call the sylvian arteries. This division of the middle cerebral artery into the ascending frontal and sylvian group is adopted solely for the purpose of convenience. While indefensible on anatomic grounds, it does permit the sure recognition of segmental arterial occlusion within the middle cerebral group. It is tempting to extend the observations to include individual arteries and it may be entirely feasible to do so;³ however, in our opinion, the variability in course and distribution of the vessels involved is too great to permit an unequivocal diagnosis.

We have observed segmental occlusion of the middle cerebral artery on the arteriograms of 42 patients with histories and neurologic findings compatible with a diagnosis of stroke; serial arteriograms were done in 22 of the cases. The ascending

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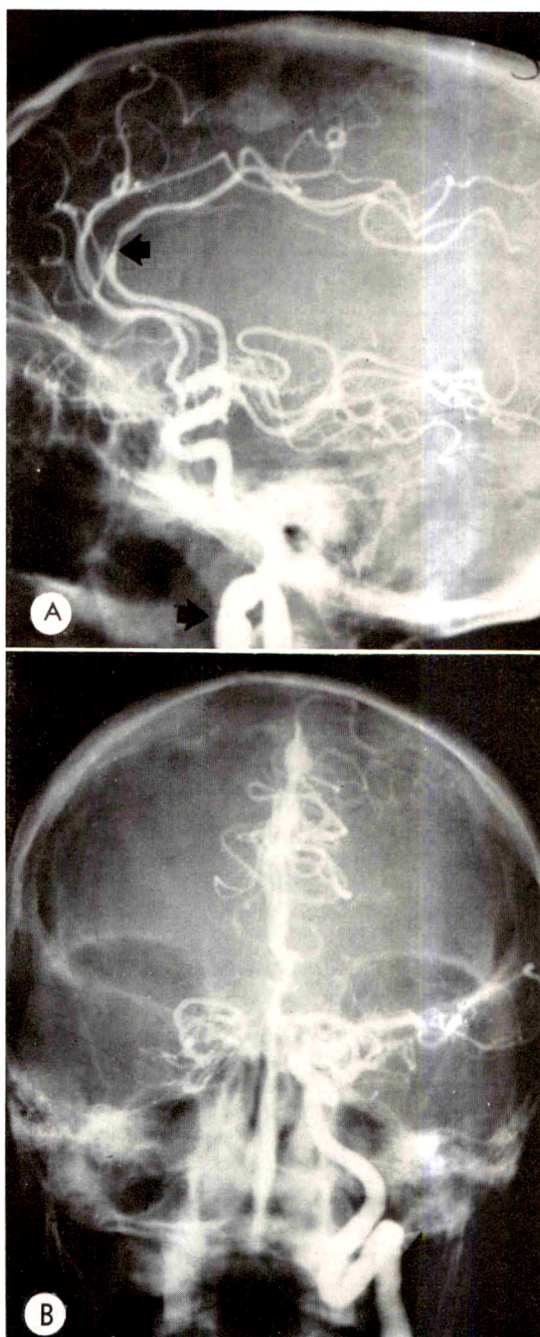


FIG. 1. (A) Occlusion of ascending frontal artery and kinking of internal carotid artery (lower arrow), with avascular zone in region of insula and frontal operculum. Upper arrow indicates stenosis of pericallosal artery. (B) Anteroposterior projection. Only the arteries in the sylvian fissure are demonstrated, with absence of vessels ascending on the insula.

frontal artery was involved in 15 patients and the sylvian group in 27. Roentgenographic features characteristic of these occlusions have already been mentioned. The diagnosis can be made from single

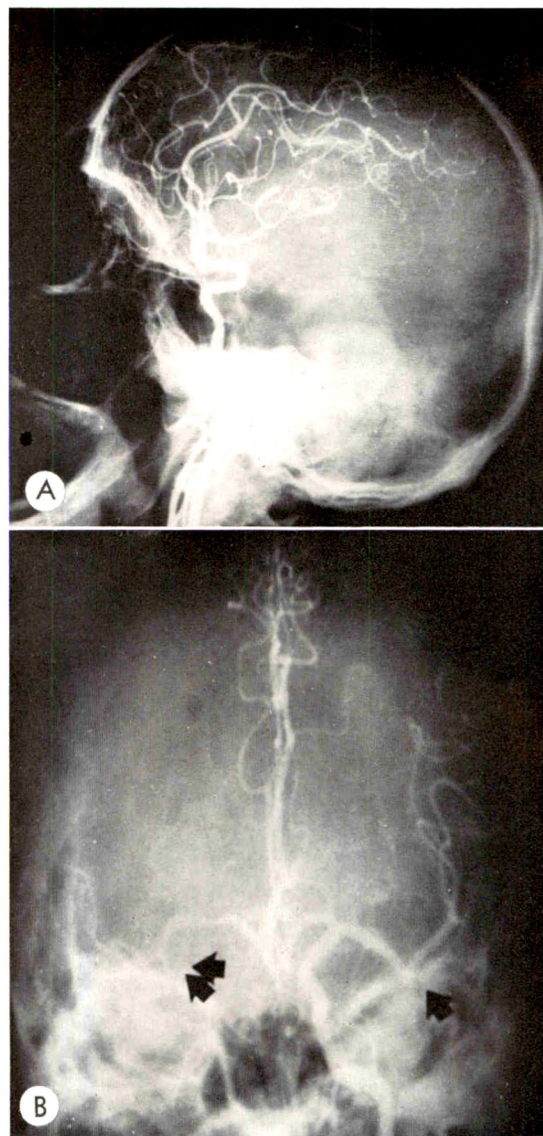


FIG. 2. (A) Occlusions of sylvian arteries. The ascending frontal artery is well demonstrated. (B) Anteroposterior projection. Double arrows indicate division of middle cerebral into ascending frontal and sylvian arteries. On the opposite side, the single arrow indicates obstruction of sylvian arteries, with filling only of the ascending frontal artery.

exposure arteriograms but becomes more certain when seriographic methods are employed, since the demonstration of retrograde filling of the proximally occluded segments from the anterior and posterior

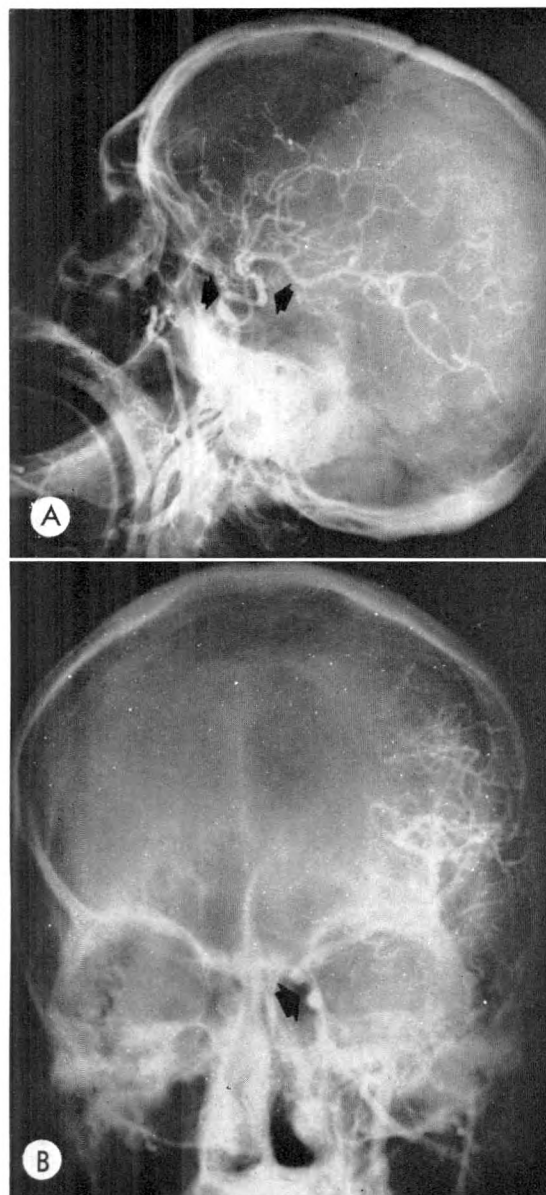


FIG. 3. (A) Patient with convulsions and focal abnormality on electroencephalogram. Arrows point to narrowed areas in left internal carotid artery. (B) Anteroposterior projection. The internal carotid artery is narrowed at arrow. Both anterior cerebral arteries are filled from the right side.

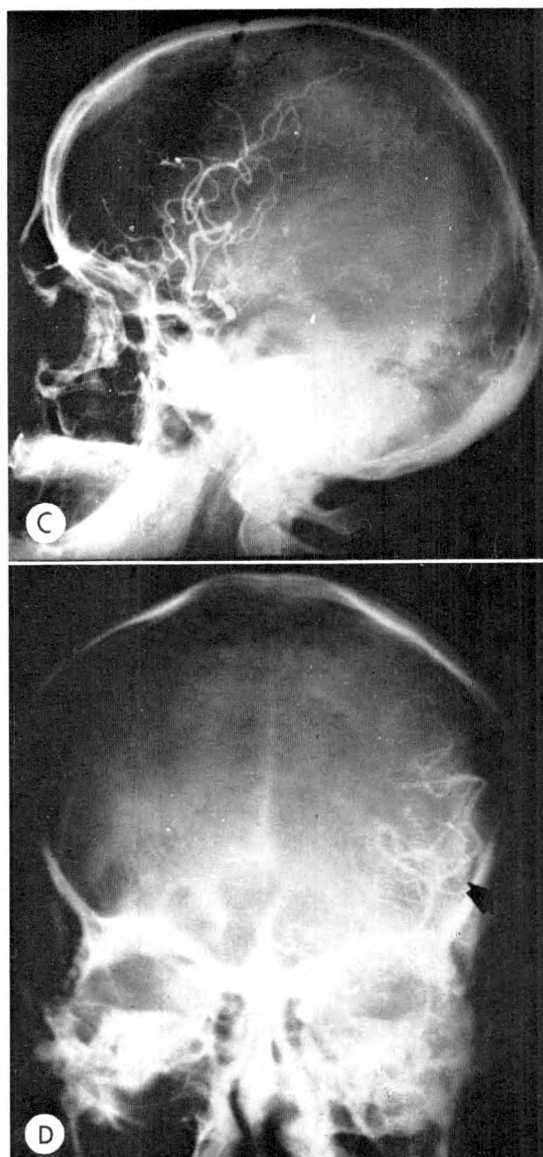


FIG. 3. (C) Same patient readmitted several weeks later with incomplete expressive aphasia and mild hemiparesis. The sylvian arteries did not fill with contrast medium. (D) Anteroposterior projection. Avascular zone at arrow.

cerebral arteries establishes the diagnosis unequivocally. Therefore, only those 22 patients in whom serial arteriography was carried out will be considered.

The neurologic abnormalities observed in these patients ranged from minimal weakness and mild mental aberration to hemi-

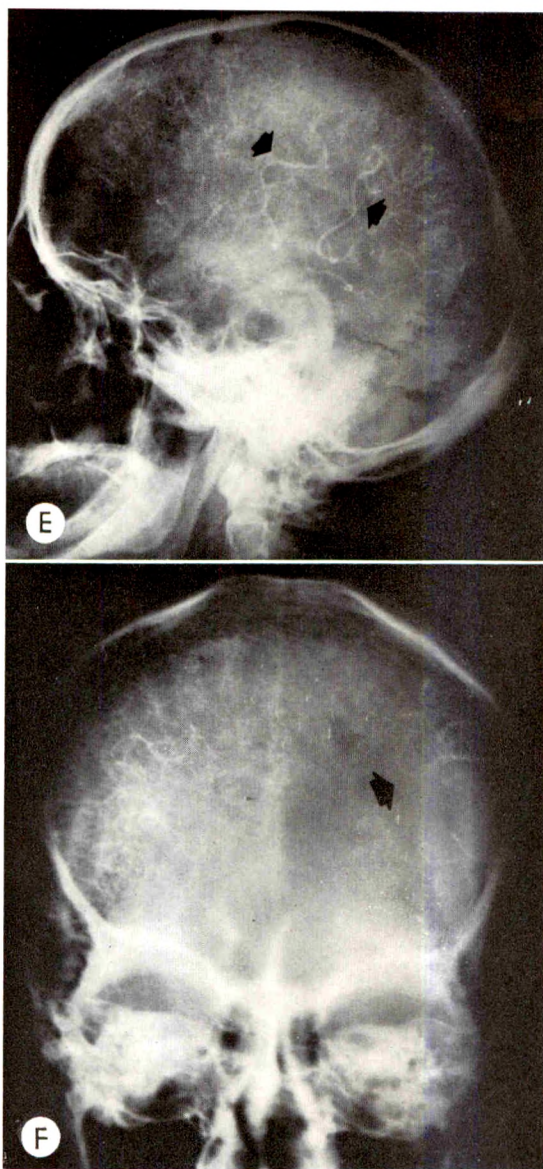


FIG. 3. (E) Right carotid arteriogram, late arterial phase. Retrograde filling of occluded segments from anterior cerebral artery (arrows). (F) Anteroposterior projection, late arterial phase. The patient improved rapidly, and after 1 week the only finding was minimal expressive aphasia.

plegia, aphasia, and coma. An exhaustive correlation between the arteriographic and clinical findings would not be valid since the neurologic examinations were performed by several different examiners. Nevertheless, the available data did per-

mit a division of the patients into 3 groups: those with severe, moderately severe, and mild neurologic deficits. Hemiplegia, marked aphasia and persistent alterations of consciousness or affect were con-

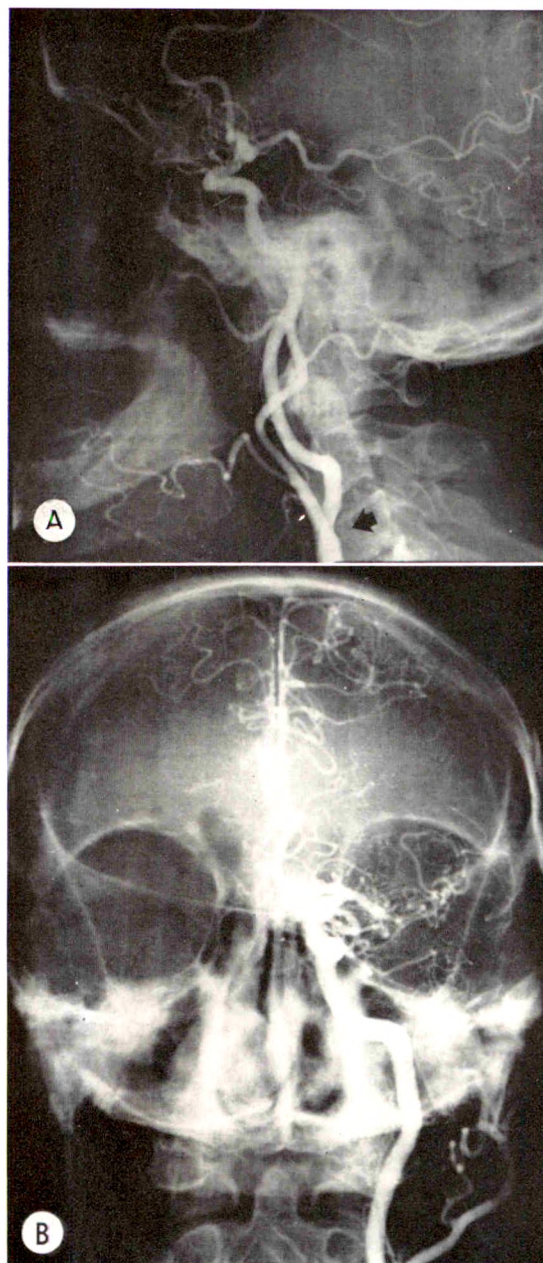


FIG. 4. (A) Occlusion of ascending frontal artery and stenosis of internal carotid artery (arrow). Patient had a mild hemiparesis. (B) Anteroposterior projection.

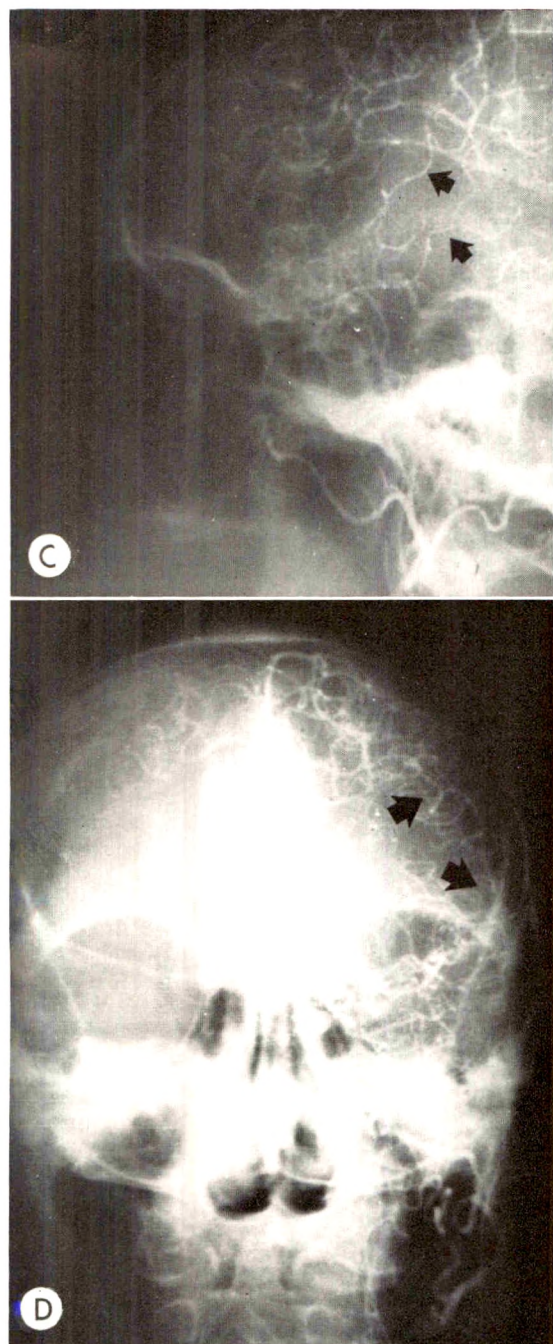


FIG. 4. (C) Same arteriogram, late arterial phase. Arrows point to retrograde filling of proximally occluded vessels. (D) Anteroposterior study, late arterial phase.

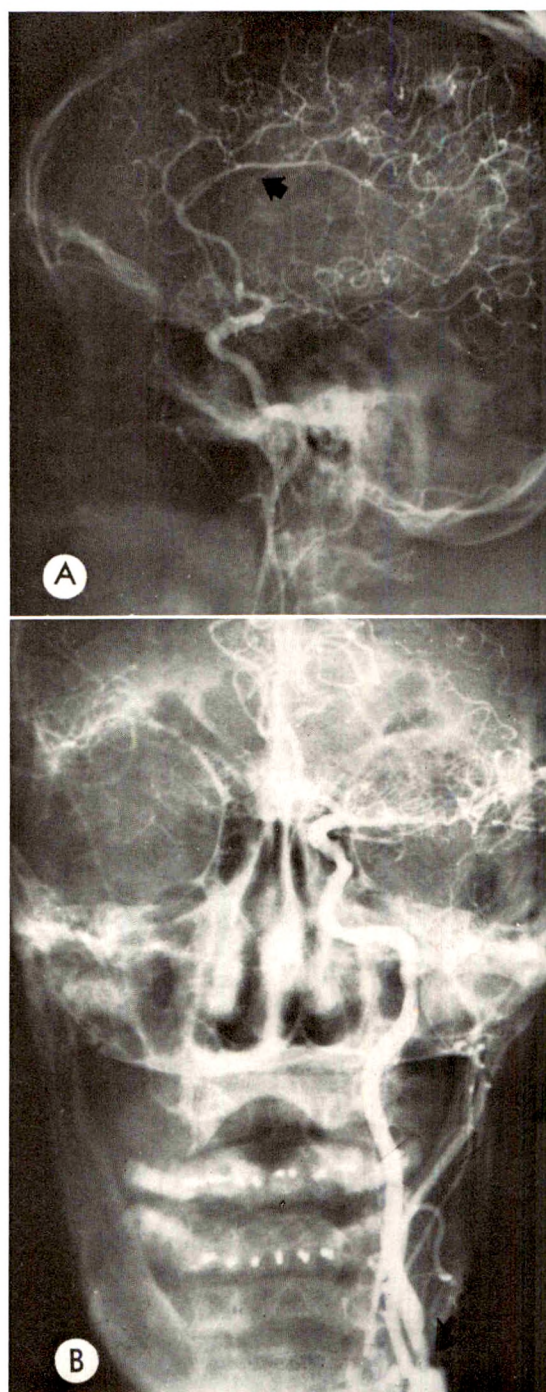


FIG. 5. (A) Occlusion of ascending frontal artery and stenosis of the internal carotid artery. Patient had a severe hemiparesis. Lateral study shows a bare area in the region of the ascending frontal artery (arrow). (B) Anteroposterior projection reveals the stenosis of the internal carotid artery near the bifurcation (arrow).

sidered to be severe neurologic deficits, patients with hemiparesis or incomplete aphasia were considered to have moderately severe disability, and those with minimal aphasia, slight changes in affect or behavior or barely detectable weakness were placed in the category of mild neurologic deficit. Of these 22 patients, the neurologic disability was severe in 10, moderate in 7 and mild in 5. Retrograde filling of the proximally occluded segments was demonstrated arteriographically in 17 patients (Fig. 3, *A-F*; and Fig. 4, *A-D*), the neurologic deficits being severe in 6 patients, moderate in 6 and mild in 5, with 4 of the 6 patients in the severe category having a history of previous stroke with marked residual impairment. Retrograde filling was not observed in 5 patients; the neurologic deficits were mild in 4 and moderate in 1, and none of these had a history suggestive of previous stroke.

Abnormalities of the extracranial portion of the carotid arteries were observed in 9 patients. Stenosis of the internal carotid near the bifurcation was found in 6 patients (Fig. 5, *A* and *B*), and kinking of the carotid artery in 3 (Fig. 1, *A* and *B*). Associated abnormalities of the anterior and posterior cerebral arteries suggestive of arteriosclerotic changes were not uncommon.

DISCUSSION

These findings indicate that segmental occlusion of the middle cerebral artery can be demonstrated arteriographically in many patients with the stroke syndrome. In some cases, the segmental occlusions are present in association with occlusive changes in other arteries, both intracranial and extracranial. In these circumstances, it is difficult to determine which lesion is responsible for the clinical picture. The issue is further complicated by the demonstration of retrograde filling of the occluded branches from the anterior and posterior cerebral arteries. The data suggest that the patients with demonstrable collateral circulation had less severe neurologic deficits

than those without arteriographically apparent collateral filling. The pial arterial anastomoses responsible for retrograde filling are small and may not be able to immediately provide a sufficient rate of flow to maintain the affected neural structures. Enlargement of the anastomoses may occur with time, but not early enough to affect the issue. However, with a slowly progressive occlusion, the collateral pathways may open up sufficiently to prevent the development of a severe neurologic deficit. This mechanism may account for the mild neurologic abnormalities observed in many of our patients with segmental occlusion and good retrograde filling.

The effectiveness of retrograde filling of proximally occluded arteries is suggested but not proved by these observations, and should be considered in an evaluation of a stroke patient. Similarly, the association of extracranial abnormalities of the carotid artery with segmental occlusion of the middle cerebral artery should not be discounted. Although the problem of stroke is complex and has many variables, the omission of complete arteriography does not appear to be a desirable simplification, for although it does not give a complete picture of the cerebral blood supply, it provides valuable information which cannot be obtained by other means. The employment of special methods, such as subtraction, might improve the effectiveness of arteriography in the portrayal of the cerebral vasculature. These findings indicate the importance of a thorough arteriographic evaluation prior to the selection, and evaluation of active therapeutic measures for the stroke patient.

SUMMARY

1. Forty-two cases of segmental occlusion of the middle cerebral artery were observed.
2. The major divisions of the middle cerebral artery are recognized as the ascending frontal group and the sylvian group.
3. Twenty-two cases were studied by

seriographic technique, demonstrating good correlation between the degree of retrograde collateral circulation from the anterior and posterior cerebral vessels and the severity of the stroke.

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VINYLITE CAST PREPARATIONS OF VARIOUS NORMAL ORGANS

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MEDICINE is constantly developing new methods by which to increase the knowledge of normal and pathologic anatomy. In recent years, one such method has been the plastic injection of blood vessels and cavities of the various organs of the body.

The first recorded attempt of an injection of any vessels was by Swammerdam in 1672.⁷ He injected the coronary arteries with hot wax stained with cinnabar. Since then a wide range of materials from dyes such as Prussian blue in aqueous solution² to injection masses such as plaster of Paris¹ have been tried without much success.

In 1907 Spalteholz and Hirsch³ used chrome yellow gelatin for injection of the coronary arteries, fixed the specimen in 10 per cent formaldehyde and bleached it with hydrogen peroxide. They then washed the specimen in water, dehydrated it with alcohol, placed it first in benzene and then in a mixture of wintergreen and benzyl benzoate, and finally into a special tank to remove all of the air and benzene. Remaining was an opaque system of vessels with a clear semitransparent heart wall. This method was one of the milestones in plastic work and provided much of the early information concerning the coronary artery circulation. However, since the clearing process was slow and incomplete, this technique was considered undesirable for future work.

The most recent and important work is the method of plastic injection followed by maceration or corrosion with acid or alkali of the specimen. Some of the most notable contributions along this line have been made by Whitten,⁹ using celloidin, Smith and Henry,⁴ using latex, and Wagner and Poindexter,⁸ using nylon for injection. However, the disadvantage of these sub-

stances is that the specimens must be kept in fluid or they will crumble or otherwise lose their shape.

Stern, Ranzenhofer and Liebow⁶ and Kazzaz and Shanklin⁵ have used vinylite for the demonstration of the coronary vessels. This material is advantageous in that, if properly used, it will outline the injected vessels and their relationships to one another, and other structures will be maintained without support. The method presented here utilizes vinylite as the injection material. The technique of Liebow and Kazzaz with certain modifications was employed. In addition, a new material, epoxy resin, was evaluated and compared with vinylite.

It was decided to inject the arteries, deep veins, and ventricles of normal human brains with red, blue, and yellow plastic material, respectively. After hardening, the brain tissue was digested away with acid or alkali, leaving a three dimensional model of the injected structures. At the beginning, we were only interested in demonstrating the relationships of the arteries and veins to the ventricular system; however, after initial success with the brain injection, this technique was applied to other organs such as the kidney (arteries and collecting system), the liver (arteries, portal and hepatic veins, and biliary tree), and the lung (arteries, veins, and bronchial tree).

MATERIAL

1. 250 ml. Ehrlenmeyer flasks
2. No. 18 intracatheter needles with plastic covering for the points
3. 1,000 ml. and 100 ml. graduate cylinders
4. Large and small glass funnels
5. "Vim" plastic, 20 ml. syringes
6. Wax paper to work on

7. Vinaseal, VYHH, from Associated Plastics, Hialeah, Florida. Epoxy resin and hardener No. 4-2-1 from Resin Coating Corporation, Miami, Florida
8. Yellow, blue and red dye (Harmon dyes from Allied Chemical Corporation, Hawthorne, New Jersey)
9. 00 silk suture material
10. Waring blender
11. Acetone
12. Normal saline
13. 20 per cent muriatic acid
14. Valspar clear varnish

METHODS

Two methods were attempted and varied only as to the plastic material used. The brains (Fig. 1, *A* and *B*) were obtained from patients who died from non-neurologic disorders. All the brains were removed carefully so as not to lacerate them anywhere. One important criterion was that the brain be fresh and not fixed. Into each of the 4 main arteries of the brain (the 2 internal carotid and the 2 vertebral arteries) No. 18 intracatheter needles were placed with plastic coverings on the pointed ends. These were tied in place securely with 00 silk. (Previously, the intracatheters themselves were tried, and not the needles, but these proved to be too narrow to allow free flow of the viscous plastic into the vessels.) At the same time another No. 18 needle was placed into the straight sinus and tied in place also with 00 silk. Through these needles normal saline was injected in amounts necessary to flush out the arterial and venous trees (measured by a lack of color to the vessels on the surface of the brain). First the arteries and then the veins were flushed, using a 20 ml. Vim plastic syringe. At this point, the brain was ready to be injected. The sequence of injection was arteries first, veins second, and ventricles third through a ventricular tap. The plastics used were viscous enough to penetrate to the arterioles and not through to the capillaries.

A. In the first method we used vinaseal, VYHH (vinylite), a poly-vinyl plastic resin that is in powder form. This was

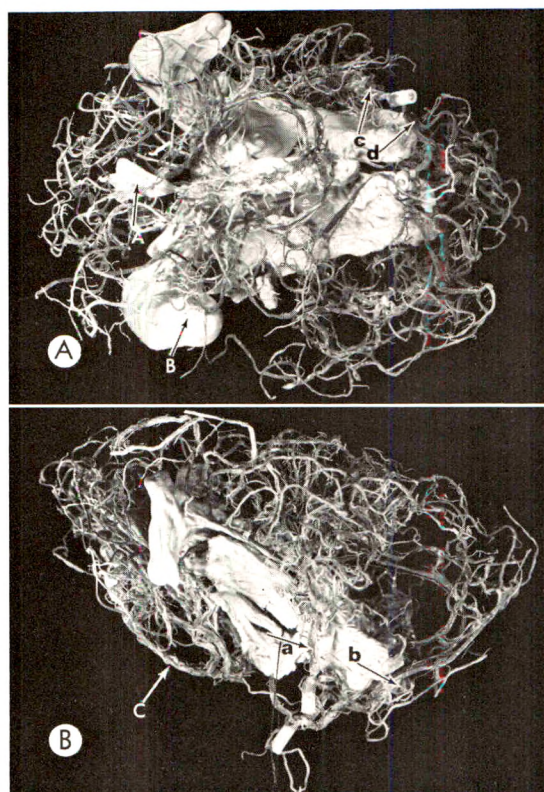


FIG. 1. (*A*) Basal view of ventricles and cerebral arteries: a—fourth ventricle; b—temporal horn; c—middle cerebral artery; d—anterior cerebral artery. (*B*) Lateral view of ventricles and cerebral arteries: a—middle cerebral artery; b—anterior cerebral artery; c—basilar artery.

mixed with acetone in a Waring blender (hard to dissolve otherwise) at room temperature, in empirical amounts, to produce a solution with a viscosity slightly greater than water. It is imperative that the blender be cleaned immediately with acetone or the plastic will be extremely hard to remove later. This material may be stored in a closed bottle for long periods of time without hardening, because hardening only occurs with evaporation of the acetone. However, once the injection of the brain is begun, it must be completed within 10 to 15 minutes or the material will harden in the vessels and in the syringes.

The arteries were injected first and tied off above the level of the needle to prevent reflux. Next, the brain was turned onto its ventral surface and the veins were injected

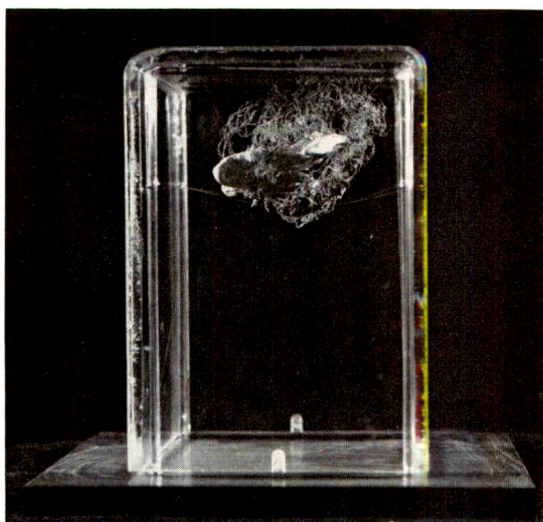


FIG. 2. Specimen mounted in case.

through the straight sinus and tied off again above the needle. A ventricular tap was done next, bilaterally, into the lateral ventricles through the dorsal aspect of the hemispheres and the plastic was injected until it was seen to emerge through the foramen of Magendie. The actual solutions for injection were made by dividing the total solution described above into 3 separate flasks and adding the respective dye to each flask and mixing well. It is best to leave off only the top of the flask of the plastic presently being used so as to prevent drying out in the other flasks.

After injection, the brain was placed in a large basin of water overnight to allow the vinaseal to harden without distortion of the brain substance that would occur if it were left to harden on a table. The next morning the brain was placed in 20 per cent muriatic acid and allowed to digest for a few days.^{1,2,5,9} The model thus formed held its shape very well, demonstrating the 3 dimensional relationships of the arteries and ventricles (Fig. 1, *A* and *B*). The model was then removed from the acid and placed in water to rinse it thoroughly. A suture was tied around the infundibular stalk and the model was removed from the water by this thread. It was allowed to dry in air for 1 to

2 hours and then sprayed with 2 coats of clear varnish (Valspar) in order to increase its hardness, and allowed to dry overnight.

The following procedure was used to mount the completed specimen: Sutures were placed around both internal carotid arteries and the basilar artery. In addition, sutures were placed through the frontal and occipital horns of both ventricles. These sutures were in turn tied to glass rods glued into a plywood base and the brain was thus suspended. A plastic cover was placed over it.

The container was made by bending 4 glass rods to assume a U shape. Eight holes were drilled in a plywood ($\frac{3}{4}$ inch thick) board to accommodate the bent glass. The glass rods were then glued into the holes and the sutures from the model were tied around them (Fig. 2).

B. The second method is similar to that described above for the epoxy resin except for the preparation of the plastic, the hardening and digestion of the brain and the fact that all the work must be done on wax paper. The epoxy resin was mixed with its hardener in a ratio of 4 parts resin to 1 part hardener, each measured in a graduated cylinder. This is necessary if good results are to be obtained. Once the two are mixed, the injection must be completed within 15 minutes or the material will harden in the syringe. The rest of the procedure was the same as in the vinaseal method until the digestion. After the injection of the epoxy resin, the brain was placed in the watertight plastic bag and a tube was tied in place at the mouth of the bag, allowing free exchange of air between the bag and atmosphere. This was then floated in a large basin of water to prevent deformity of the brain. The reason for doing this is that the epoxy, unlike the vinaseal, will not harden in water, but rather needs air in order to harden. It was left like this for 10 days to harden and then the brain itself was placed in a basin of water for 2 to 3 weeks for maceration of the brain substance. The epoxy is soluble, as is the vinaseal, in ace-

tone and therefore copious amounts should be on hand to clean up any spillage.

In addition, adult lungs (Fig. 3, *A* and *B*) and infant lungs (Fig. 4, *A* and *B*) (unexpanded bronchial system, arteries and veins); kidneys (Fig. 5, *A* and *B*) (collecting system and arterial tree); and liver (Fig. 6, *A* and *B*) (hepatic vein and artery, biliary tree and portal vein) were injected in a similar manner using only the vinaseal method. The models thus produced held their shape very well. The material penetrated to the terminal arterioles, venules, bronchioles, alveoli, and biliary radicles.

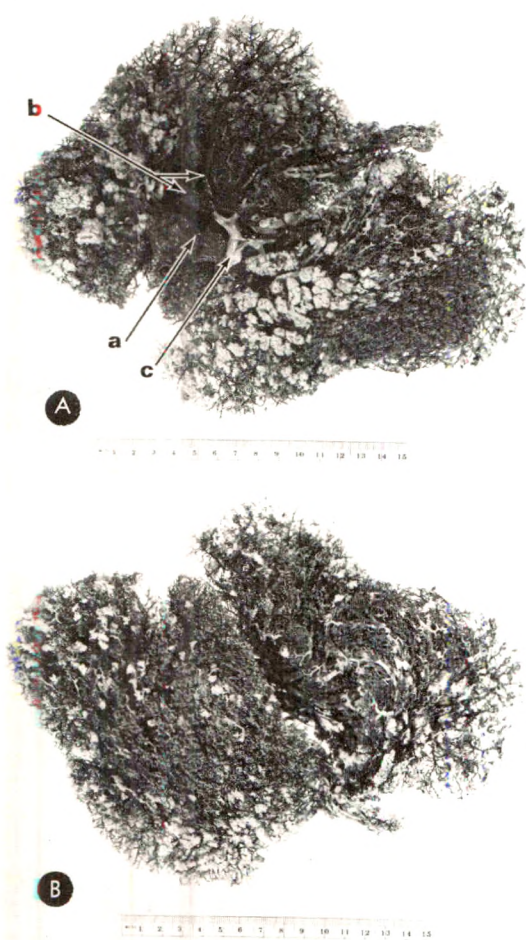


FIG. 3. (*A*) Medial view of arteries, veins, and bronchial tree of adult lung: a—pulmonary artery; b—pulmonary vein; c—main stem bronchus. (*B*) Lateral view of adult lung.

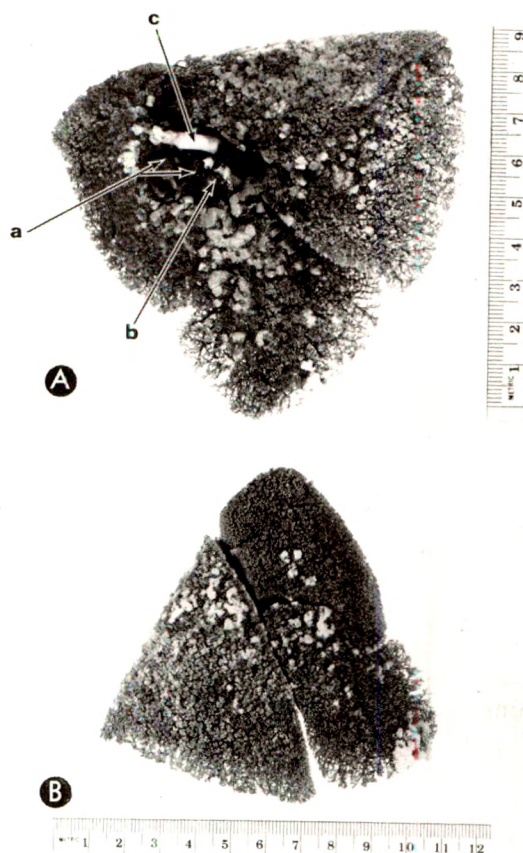


FIG. 4. (*A*) Medial view of arteries, veins, and bronchial tree of infant lung: a—pulmonary artery; b—pulmonary vein; c—main stem bronchus. (*B*) Lateral view of infant lung.

For selective evaluation of just one of these systems in any organ, an injection of only that system can be made, leaving the other channels uninjected.

RESULTS

Using both methods, the arteries were injected to their terminal arterioles. However, in the epoxy-injected brain, these vessels did not retain their anatomic relationship to one another due to collapse. For demonstration, they were placed flat on a white cardboard and labeled. Arteries were much firmer and easier to work with when injected with the polyvinyl plastic. The arteries thus injected held their normal anatomic shape very well. The ventricles also

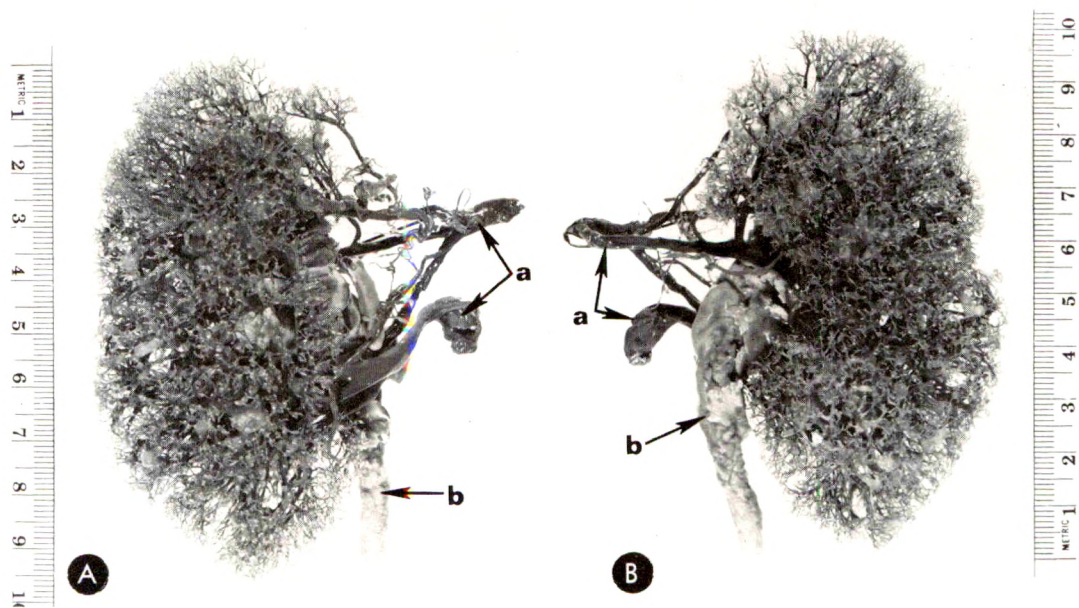
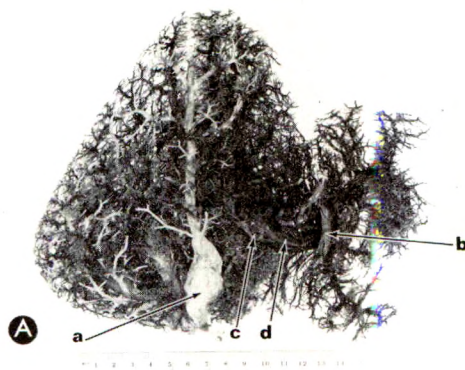


FIG. 5. (A) Anterior view of arteries and collecting system of adult kidney: a—renal artery; b—ureter. (B) Posterior view of adult kidney: a—renal artery; b—ureter.



held their shape well with the vinaseal, but did not visualize at all with the epoxy. The deep venous system did not visualize with either method due to poor removal of the dura. In all the other organs, the injected material held its shape well with the vinaseal.

SUMMARY AND CONCLUSION

Our initial interest in proceeding with this project was to make some 3 dimensional models of the brain showing the relationships of the arteries, veins, and ventricular system to use as a teaching aid in neuro-radiology. We had hoped also to inject the



FIG. 6. (A) Posterior view of hepatic artery and vein, portal vein, and biliary tree: a—hepatic vein; b—hepatic artery; c—bile duct; d—portal vein. (B) Anterior view of liver.

subarachnoid cisterns but could not get permission to inject this area because the injection had to be carried out before the brain was removed from the body and, therefore, there might be leakage into the face with subsequent discoloration.

After our initial success with the injection of arteries, veins and ventricles of brains, we tried to find an injection material that would be so rigid after injection that 3 dimensional relationships would be maintained. We evaluated epoxy resin and found it inferior to vinaseal.

We then extended our project to include various other organs. Liver, kidneys, and lungs proved easier to work with than brains and the specimens were excellent.

We plan to extend our project to the study of collateral circulation pathways in intracerebral thrombosis and vascular patterns in malignant brain tumors. The method can easily be adapted to the study of altered architecture in other organs with various pathologic conditions.

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DARKROOM PRACTICE AND UNNECESSARY PATIENT EXPOSURE*

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IT IS well known that darkroom practice has a substantial influence on the exposure required to produce a roentgenogram whose densities fall within the useful range of film blackening. If development time is too short, developer temperature too low, or if the strength of the developer is allowed to become depleted, more exposure is required than when proper darkroom techniques are employed. Since this added exposure falls within the realm of unnecessary patient exposure, radiologists must be concerned with it. Indeed, the need for concern is doubly great because the same darkroom practices which create unnecessary patient exposure also produce degradation in the contrast and informational content of films.

In an effort to determine the extent to which darkroom practice is currently leading to excessive patient exposure, a study of the roentgenographic darkrooms currently operated by physicians and by hospitals within the city of Baltimore has been undertaken. Test films were given a series of graded exposures and submitted to the several darkrooms with the request that they be processed in a manner consistent with normal darkroom technique. After processing, the films were examined densitometrically and the exposures in milliroentgens required to produce a film density of 1.0 above fog (the mid-point in the useful range of film blackening) were determined. The values so obtained were then compared with the exposures needed to produce in these films a similar density under optimum darkroom conditions.

DARKROOM SELECTIONS

The darkrooms selected for this study were those maintained in hospitals and in

TABLE I
CLASSIFICATION OF DARKROOMS STUDIED

Class of Darkroom	No. Studied
Radiologists' Offices	12
Other Physicians' Offices	18
Hospitals: (a) Hand-operated	7
(b) Automatic	12
All Classes	49

physicians' offices which contained diagnostic apparatus registered with the Maryland Department of Health. Since the number of darkrooms located within the corporate limits of Baltimore was substantial, no darkrooms in the metropolitan area beyond these limits were studied. Also, not included were those darkrooms for which Health Department records indicated a weekly case load of 10 patients or less; these were considered to have an insufficient impact on the problem of unnecessary patient exposure to merit study. Several other darkrooms were also excluded because they had not been registered with the Health Department at the time of the study. However, the number of these was small.

In all, 49 darkrooms were studied. These included 19 darkrooms in 12 general hospitals, 12 darkrooms in radiologists' offices, and 18 darkrooms in the offices of other physicians (Table 1). Of the hospital installations, 12 were of the automatic processing type, whereas 7 employed so-called hand processing methods. All of the physician installations, both radiologist and non-radiologist, were of the hand operated variety. The cooperation provided by both hospitals and physicians in this study was excellent. In only one instance was there

* From the Department of Radiological Science, The Johns Hopkins University and the Department of Health, State of Maryland. The work described in this paper was supported in part by U. S. Public Health Service Contract SApH 76496.

TABLE II
DISTRIBUTION OF DARKROOMS CLASSIFIED ACCORDING TO EXPOSURE
REQUIRED TO PRODUCE DENSITY OF 1.0 IN TEST FILMS

Class of Darkroom	Exposure (mr)									
	1.2- 1.4	1.4- 1.6	1.6- 1.8	1.8- 2.0	2.0- 2.2	2.2- 2.4	2.4- 2.6	2.6- 2.8	2.8- 3.0	3.0- 3.2
Radiologists' Offices	4	4	2	1	0	0	0	1	0	0
Other Physicians' Offices	7	4	3	1	0	1	0	1	0	1
Hospitals:										
(a) Hand-operated	2	2	2	0	1	0	0	0	0	0
(b) Automatic	1	5	1	3	0	1	1	0	0	0
All Classes	14	15	8	5	1	2	1	2	0	1

reticence on the part of a hospital to participate.

EXPERIMENTAL PROCEDURE AND RESULTS

Eight by ten inch sheets of a widely used brand of radiographic film (DuPont Cronex I) were each subjected to a series of graded exposures with radiation having a half value layer of 2 mm. Al. In all cases, exposures were made with intensifying screens (DuPont Par Speed) and with an exposure time of 0.1 sec. Radiation was provided by a constant potential x-ray generator whose beam intensity was maintained well within ± 1 per cent of a preset level. Exposure values were varied by changes in the distance between film and x-ray tube target in accordance with the inverse square law after appropriate correction for air attenuation.

After exposure, the films were processed through the several darkrooms included in the study. In this phase of the work, every effort was made to ensure that the film development procedures used by each darkroom followed as closely as possible the techniques conventionally employed. Often, a darkroom operator was unaware that a test film was included among the films being developed.

After processing, the films were examined densitometrically and the exposures

required to produce a radiographic density of 1.0 above fog were determined.

Exposure values ranged from 1.25 to 3.20 mr. Table II gives the distribution in the number of darkrooms, classified according to test film exposure value.

Table III lists the average exposure levels, in milliroentgens, required to produce a film density of 1.0 for each of the types of darkroom studied. It will be observed that the average exposure ranged from 1.55 mr for hand-operated hospital darkrooms to 1.75 for automatic hospital darkrooms.

Darkroom performance was also measured in terms of the density versus log exposure gradient observed at a density of 1.0 in the test films. Since the informational content of a radiographic film is a function of this gradient, these measurements pro-

TABLE III
AVERAGE EXPOSURE REQUIRED TO PRODUCE
DENSITY OF 1.0 IN TEST FILMS

Class of Darkroom	Average Exposure (mr)
Radiologists' Offices	1.60
Other Physicians' Offices	1.70
Hospitals: (a) Hand-operated	1.55
(b) Automatic	1.75
All Classes	1.65

TABLE IV
DISTRIBUTION OF DARKROOMS CLASSIFIED ACCORDING TO DENSITY
VERSUS LOG EXPOSURE GRADIENT OF TEST FILMS

Class of Darkroom	Density Versus Log Exposure Gradient (contrast factor)								
	1.1-	1.3-	1.5-	1.7-	1.9-	2.1-	2.3-	2.5-	2.7-
	1.3	1.5	1.7	1.9	2.1	2.3	2.5	2.7	2.9
Radiologists' Offices	0	0	1	0	1	3	2	4	1
Other Physicians' Offices	1	1	0	2	2	1	5	5	1
Hospitals: (a) Hand-operated	0	0	0	0	2	2	1	2	0
(b) Automatic	0	0	0	0	1	2	2	5	1
All Classes	1	1	1	2	6	9	10	16	3

vided an estimate of the quality of the films that may be expected from the darkrooms in question. Table IV gives the distribution of the several darkrooms classified according to the observed values of this gradient. Table V lists average values of the gradient as well as average values of the background fog density observed.

DISCUSSION

The data presented in the foregoing tables are of interest from several points of view. With reference to Tables II and III, it will be noted that on average the performance characteristics of the several classes of darkrooms studied are quite similar. The best performance occurred in hospital darkrooms with hand-operated equipment where the test films required an average exposure of 1.55 mr to yield a film density of 1.0. Close behind were the darkrooms in radiologists' offices, followed by the dark-

rooms of the offices of other physicians and the automatic darkrooms of hospitals. The similarity in darkroom characteristics from class to class is noteworthy in view of the rather wide spread observed in the exposure values of individual darkrooms.

A note is perhaps in order concerning the relatively poor performance of the automatic processing equipment. This finding is possibly more apparent than real. It is well known that in darkrooms equipped with hand-operated apparatus, films are frequently inspected during processing. If the film has been overexposed, its development time is shortened in order that the included densities may fall within the useful range of film blackening; that is, films may be processed for shorter times than that recommended. Darkroom technicians in this study, on the other hand, generally developed the test films without inspection and for recommended processing times. Hence, the exposure values shown in Table III for hand-operated equipment are possibly lower than those which prevail in practice. It also should be pointed out that the performance rating for automatic darkroom equipment shown in Table III is by no means an inherent characteristic of such apparatus. Indeed, one may operate automatic equipment to yield results which range from little to almost complete development of the film.

It is of interest to report that when the test films used in this study are fully de-

TABLE V
AVERAGE D-LOG E GRADIENT AND BACKGROUND
FOG DENSITY OF TEST FILMS

Class of Darkroom	D-log E Gradient	Fog Density
Radiologists' Offices	2.4	0.10
Other Physicians' Offices	2.2	0.10
Hospitals: (a) Hand-operated	2.3	0.11
(b) Automatic	2.4	0.09
All Classes	2.3	0.10

veloped, an exposure of only 1.0 mr is required to produce a radiographic density of 1.0 above fog. Hence, from the data shown in Tables II and III, it appears that on average, approximately 65 per cent more radiation than necessary is being currently used in the city of Baltimore for the production of diagnostic roentgenograms because of incomplete film processing; *i.e.*, if all films were developed to completion or reasonably close to this point, patients would be exposed to only three-fifths of the radiation presently employed. There are some who perhaps may argue that the processing of roentgenographic films to completion is impractical. Under such circumstances, a darkroom technician has no opportunity to compensate for accidentally underexposed films by means of overdevelopment. Also, some may express concern lest complete development introduce excessive amounts of background fog. The first argument has little or no substance. It has been estimated by film suppliers that 75 per cent of the film exposed in Baltimore is currently processed through automatic development apparatus. Under such circumstances, an operator has no chance of correcting errors of exposure with alterations in darkroom technique. As for excessive fog density, observations indicate that this need not be a serious problem. Table VI lists the fog densities observed in the test films of this project, tabulated according to the corresponding exposure values required to produce a film density of 1.0 above fog. It is evident that these densities increased only slightly as an exposure value of 1.0 mr was approached. A greater increase might have been noted, of course, if the test films had not been reasonably fresh. However, the use of anything but fresh film is undesirable on many counts. Hence, there is little question that every effort should be made by radiologists and other physicians to alter their darkroom practices so that more complete development of films is attained. By this step, a substantial reduction in unnecessary ex-

TABLE VI
FOG DENSITIES OBSERVED IN TEST FILMS*

Average Fog Density	Exposure (mr)
0.107	1.2-1.4
0.104	1.4-1.6
0.096	1.6-1.8
0.084	1.8-2.0
0.089	2.0-3.2

* Tabulated according to corresponding exposure values required to produce film density of 1.0 above fog. Decreasing exposure values are indicative of increasing levels of film development.

posure of patients may be achieved.

The benefits of improved darkroom practice are by no means limited to reduced patient exposure. Measurements of the density versus log exposure gradient of the test films used in this study indicate that when these films are processed to completion, the resulting gradient approaches a value of 2.9. Such a value represents a substantial improvement over those currently prevailing in non-radiologists' offices and a not inconsiderable improvement over the values obtained in many installations under radiologist control (see Tables IV and V).

It should be emphasized that the data obtained from the City of Baltimore are indicative only of the darkroom practices of one large Eastern city of the United States. However, there is little reason to believe that the Baltimore experience may be widely different from that observed elsewhere. Hence, increased attention toward the improvement of darkroom practice seems in order. Certainly, the darkroom is one place where techniques may be altered to reduce patient exposure.

SUMMARY

A study of the practices of 49 darkrooms associated with hospitals and physicians' offices in the city of Baltimore has been undertaken to determine the extent to which these practices contribute to unnecessary patient exposure. The darkrooms studied included 12 in radiologists' offices,

18 in the offices of other physicians and 19 in general hospitals. Of the latter, 7 used hand-operated equipment, whereas 12 employed so-called automatic processing apparatus. The performance characteristics of all classes of darkroom, on average, were similar. From knowledge of the exposure characteristics of the test films when developed to completion, it is estimated that

improvements in darkroom technique could reduce patient exposure by approximately 40 per cent. The diagnostic quality of the processed films also would be bettered by these changes.

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Representative on the Board of Chancellors of the American College of Radiology: Seymour F. Ochsner.

Sixty-sixth Annual Meeting: Washington Hilton Hotel, Washington, D. C., Sept. 28-Oct. 1, 1965.

AMERICAN RADIUM SOCIETY

President: Joseph H. Farrow, New York, N. Y.;
President-Elect: Justin J. Stein, Los Angeles, Calif.; *1st Vice-President:* Robert C. Hickey, Madison, Wis.; *2nd Vice-President:* Marvin Williams, Rochester, Minn.; *Secretary:* John L. Pool, New York, N. Y.; *Treasurer:* Milton Friedman, 1016 Fifth Ave., New York 28, N. Y.

Executive Committee: Robert L. Brown, Chairman; Gilbert H. Fletcher; Charles G. Stetson; Joseph H. Farrow; John L. Pool; Justin J. Stein; Robert C. Hickey; Marvin Williams; Milton Friedman.

Scientific Program Committee: Joseph H. Farrow, Chairman; Juan del Regato, Colorado Springs, Colo.; Roald N. Grant, New York, N. Y.; Robert C. Hickey, Madison, Wis.; John H. Maus, Windsor, Ont.; Sidney Rubinfeld, New York, N. Y.; Charles G. Stetson, Tenafly, N. J.; Justin J. Stein, Ex officio.

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Publication Committee: Harry Hauser, Chairman, Cleveland, Ohio; Wendell C. Hall, Hartford, Conn.; Martin Van Herik, Rochester, Minn.

Public Relations Committee: John Day Peake, Chairman, Mobile, Ala.; H. Peter Mueller, Belmont, Mass.; James M. Ovens, Phoenix, Ariz.

Janeway Lecture Committee: Edith H. Quimby, Chairman, New York, N. Y.; William S. MacComb, Houston, Tex.; Clifford L. Ash, Toronto, Ont.

Representatives on the American Board of Radiology: Justin J. Stein, Los Angeles, Calif.; Bernard P. Widmann, Philadelphia, Pa.; Donald S. Childs, Jr., Rochester, Minn.

Representatives on the National Committee on Radiation Protection: Robert Robbins, Philadelphia, Pa.; Herbert M. Parker, Richland, Wash.

Representative on the Board of Chancellors of the American College of Radiology: Charles G. Stetson.

Forty-seventh Annual Meeting: Roosevelt Hotel, New Orleans, La., April 8-10, 1965.

EDITORIALS

THE CARDIOVASCULAR RADIOLOGIST

OF THE various methods available for the diagnosis of congenital cardiac malformations in infancy and childhood, angiocardiology has assumed a prominent role. Cardiac catheterization, in its classic form, consisted of exploring every compartment within the heart with a "physiologic" catheter. After several hours the result was a weary patient and yards of records with a myriad of curves, oscillations, and wiggly lines. In the final analysis, an anatomic diagnosis was constructed using physiologic bricks.

With the development of rapid filming techniques and the use of safe contrast material, an entirely new dimension was added. Despite the obvious advantages and the wide acceptance of angiocardiology, there is still a tendency to follow the classic physiologic approach and then add angiocardiology to the procedure just to be certain that the physiologically constructed anatomy is, in fact, present.

Practically all congenital cardiac malformations are gross enough to allow precise visualization by angiocardiology. The congenital heart in this respect is no different than any other organ in the body which is grossly altered by congenital malformation. Who would consider, for example, attempting to diagnose a congenital anomaly of the urinary tract by physiologic methods? Is it not reasonable to reverse the classic diagnostic approach and attempt to shorten the duration of the entire procedure by performing the angiocardiology at the outset? It seems logical to secure the anatomic diagnosis initially and then selectively obtain those physiologic studies which will point up specifically the nature and the degree of hemodynamic

alteration resulting from the known malformation.

From a strictly practical point of view, many of the physiologic studies are only complementary in the face of a precisely demonstrated malformation. Nadas,¹ for instance, expresses the opinion that "perhaps the most important, and in many ways the most accurate information obtained by the cardiac catheterization procedures is the measurement of pressures." These measurements are probably best obtained prior to the final placement of the catheter for the angiocardigraphic studies since the contrast material may alter intracardiac pressures. In selected cases, some method may be required to determine the ratio of systemic to pulmonary flow.

This viewpoint implies that there is an anatomic or diagnostic phase and a physiologic or evaluative phase to the procedure. Another implication is that the diagnostic phase of the procedure ideally belongs in the hands of the cardiovascular radiologist. As Taveras² so aptly states "when faults of technique are committed by someone else, misinterpretation often follows. The head must always know what the hands have done."

If the pediatric cardiologist approaches the angiocardigraphic phase of the procedure with the same understanding of roentgenographic and angiocardigraphic principles as the cardiovascular radiologist, then there is no useful purpose in having the radiologist assume the responsibilities of the actual procedure. Often, however, this approach is not observed and, as a re-

¹ NADAS, ALEXANDER S. *Pediatric Cardiology*. Second Edition. W. B. Saunders Co., 1963, p. 159.

² TAVERAS, JUAN M., and WOOD, ERNEST H. *Diagnostic Neuroradiology*. Williams & Wilkins Co., 1964, Preface.

sult, studies of optimum quality are not consistently obtained. It is also true that when the radiologist is not actively involved in the procedure and has a "laissez faire" attitude about it then the radiology technicians may also reflect this attitude and poor quality work is the end result. There is too much human effort, time and expense involved in cardiac diagnostic procedures to allow anything less than an optimum angiocardigraphic study on practically every occasion.

In the past, it is true, the radiologists were not equipped to accept the responsibilities of cardiac diagnostic procedures. However, the specialty is undergoing a radical change. In recent years with the development of cardiovascular radiology, a new group of radiologists has appeared on the scene. Many of these young men are seeking out cardiovascular training programs where they may learn the techniques and principles of angiocardigraphic diagnosis; such programs should also include a knowledge of the electrocardiographic manifestations of cardiac arrhythmias and the indications and use of cardiac resuscitative measures.

With the increasing role of angiocardiology in the diagnosis of cardiac malformations, it is only reasonable to assume that the trained cardiovascular radiologist would want to be involved actively in these procedures and be able to and would want to assume the responsibilities entailed. Anyone familiar with this field could cite numerous examples of congenital cardiac malformations where the diagnosis and the evaluation rest almost entirely on precise angiocardiology.

For the practical minded surgeon, there is no substitute for the visual demonstration of grossly altered anatomy prior to surgery. For the younger, less experienced surgeon, it provides a sense of security and also allows him to think specifically about the lesion that awaits him. Well performed angiocardiology and precise filming techniques enhance the service to the patient and are a delight to the diagnostic radiologist who is vitally interested in the art of demonstrating a hidden anatomic structure.

LEWIS S. CAREY, M.D.

Saint Joseph's Hospital
Saint Paul 2, Minnesota





HENRY JANNEY WALTON, M.D.
1879-1965

DR. HENRY J. WALTON died after a short illness on January 24, 1965 only 10 days following his 86th birthday. His death at the Union Memorial Hospital, Baltimore, Maryland, was due to a ruptured abdominal aortic aneurysm. His passing further thins the ranks of distinguished pioneer radiologists. At the time of his death, he was Professor Emeritus of Radiology at the University of Maryland School

of Medicine, having retired as Professor of Radiology and Director of the Department of Radiology of the University Hospital in 1945 after 30 years of devoted service to a specialty he loved and to which he made a lasting contribution. His retirement from hospital and medical school work gave him added interest and time for the private practice of radiology. For the next 10 years he was able to enjoy practicing clinical

radiology and to continue to put into practice certain principles which he strongly believed would elevate the specialty from laboratory radiology to a level on par with other branches of clinical medicine.

Dr. Walton was born on January 14, 1879 in Genoa, Nebraska, where the Maryland family had moved to develop a cattle ranch. He returned to Maryland with his family in 1893, where he received his early education at Friends School and Baltimore City College.

Following his graduation from the Baltimore Medical College in 1906, an institution which later merged with the Physicians and Surgeons Medical College to become the University of Maryland Medical School, he interned at Mercy Hospital here in this city. He immediately began the practice of medicine and surgery; however, his interest in roentgenology led to his appointment as assistant to Dr. Henry Chandlee, the roentgenologist of the University of Maryland Hospital. More and more of his time was confined to this relatively new and fascinating modality of diagnosis and treatment. Feeling the need for more training, Dr. Walton spent 2 years with Dr. Frederick H. Baetjer, Chief Radiologist at the Johns Hopkins Hospital. Upon the death of Dr. Chandlee in 1916, Dr. Walton was made head of the Department of Roentgenology of the University of Maryland Hospital.

Dr. Walton spared no time or effort in the struggle to develop and expand his department and was always years ahead of his hospital administrators in ideas and plans. His gratitude for having lived long enough to see his department grow from one room and a closet to one of several wings and floors, an outstanding department, was evidenced in his establishment of a lectureship in radiology in 1960 at the University of Maryland Medical School. Dr. Walton was a strong believer in limiting work to that which could be done well, in practicing clinical radiology with as much knowledge about a patient as possible at hand when making his reports, and

in discouraging the practice of blind "film reading" at every opportunity. His interest in obstetrical radiology and the development of a method of pelvimetry will be remembered. The distortion chart developed 30 years ago is still being used today. Dr. Walton strived to make his department complete and all inclusive and resented the invasion by other specialists who would insist on doing part of the radiological study that the radiologist should do himself.

Dr. Walton was a man of gentle habits, impeccable character, charming personality and immaculate dress; one who was easy to know, willing to listen and ready to extend a hand with a hearty greeting. He was faithful to his church, family and club. His charming wife added to a home atmosphere which left little to be desired and which was deeply reflected in his children and grandchildren.

Dr. Walton was a member of the Baltimore City Medical Society, the Medical and Chirurgical Faculty of Maryland, the Southern Medical Association, the American Medical Association, and the American Roentgen Ray Society.

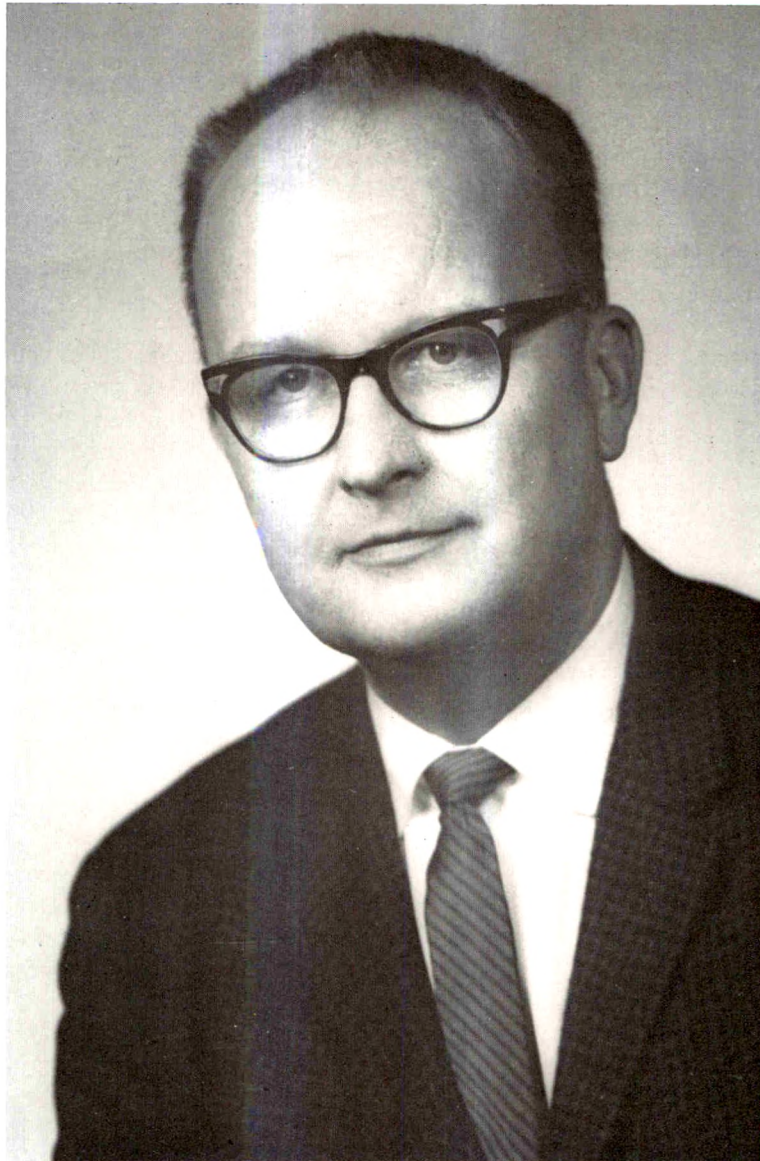
He was past chairman of the radiological sections of the Baltimore City Medical Society and of the Southern Medical Association, charter diplomate of the American Board of Radiology, past president, charter member and fellow of the American College of Radiology.

Dr. Walton is survived by his wife, Helen Smith Walton; a son, William E. Walton of Atlanta, Georgia; 2 daughters, Mrs. Richard Manning of New Canaan, Connecticut, and Mrs. DeLanson Lenhart of Riverside, Connecticut; and 7 grandchildren.

A reverent salute is extended to a former associate, a man of dedicated interest in his specialty and of noble character, by the writer and his partners, Dr. Charles N. Davidson, Dr. Henry H. Startzman, Jr., and Dr. Rawdon E. Rambo.

WALTER L. KILBY, M.D.

115 Medical Arts Building
Baltimore, Maryland 21201



LEO A. NASH, M.D.
1910-1964

WITH the sudden death of Leo A. Nash on December 1, 1964, many of us lost a dear and cherished friend and medicine an exceptionally competent and skilled radiologist.

He was born on June 26, 1910, in Chisholm, Minnesota, and received his undergraduate training at the University of

Minnesota. Also receiving his medical training there, he obtained the degree of Doctor of Medicine in 1934. After a year's internship in pediatrics at the University of Minnesota, he served a rotating internship at the Cleveland City Hospital in 1935-1936. Following this, he was in the private practice of medicine for 18 months in River

Falls, Wisconsin, being associated with Dr. Charles Dawson. Strongly impressed with the great advances made in the care of patients as the result of radiologic procedures, he obtained a fellowship in radiology at the Mayo Clinic, where he was trained from January 1, 1938, to January 1, 1941. During this period he had the good fortune to be closely associated with such outstanding men in the specialty as Drs. B. R. Kirklin, John D. Camp, Harry M. Weber, and Charles G. Sutherland. He was especially proud of the strong friendship that he acquired with Dr. Eugene T. Leddy during that period. Toward the end of his fellowship at the Mayo Clinic, he was awarded the degree of Master of Science in Radiology by the Mayo Foundation of the University of Minnesota and also in 1941 he was certified as a diplomate by the American Board of Radiology.

From January, 1941, to June, 1944, he was in Fargo, North Dakota, where he was radiologist to St. John's Hospital. In June, 1944, he was asked to become associated in the practice of radiology with Drs. Edward Schons, Harold O. Peterson and John Paul Medelman in St. Paul, Minnesota. As a member of this group, he was active in their office and was radiologist at St. Joseph's Hospital for several years. In recent years he had been the head of radiology at St. Luke's Hospital and consultant at Riverview and Children's Hospitals. A clinical instructor in radiology at the University of Minnesota, he was especially concerned with the instruction and welfare of the interns as well as with the development and expansion of the radiologic departments with which he was associated, and had much to do with the installation of the best and most modern radiologic equipment available for the better care of the patients in these departments.

Not one to seek aggrandizement, he was still ever active in the societies to which he belonged. These included the American Roentgen Ray Society, the Radiological Society of North America, and the American College of Radiology. From 1949 to

1951 he was secretary-treasurer of the Minnesota Radiological Society, and in that capacity he was unusually active and accepted many responsibilities not ordinarily an obligation of one in that position. In 1957 he was vice-president and in 1958 president of the Minnesota Radiological Society. During these years he was acutely aware of the problems regarding the economic aspects of radiology as they affected not only the physician but also the patient. As a result, he worked industriously with others in attempts to improve contracts with Blue Cross and Blue Shield and provide greater and more equitable benefits and payments.

Counselor for the Minnesota Radiological Society to the American College of Radiology in 1954, 1955, and 1956, he subsequently served on the Minnesota Radiological Society Nominating Committee for Fellowship in the American College of Radiology (in 1962, 1963, and 1964). In his affiliations with the Minnesota State Medical Association and the various radiologic societies, he always gave much in the way of wise and prudent counsel, and his kindness and selfless effort in this regard was only tempered at times by his impatience with those he felt were lagging in their efforts or timid in their support. In later years he became solicitous of the welfare of elderly physicians in Minnesota who had encountered financial difficulties after they had retired from the practice of medicine because of age or illness, and consequently he devoted much of his time to this cause as a member of the Physicians' Assistance Committee of the Minnesota State Medical Association and the Ramsey County Medical Foundation for Aid to Indigent Physicians.

We who knew Leo well will long remember him for his unique and refreshing sense of humor, his great faith in his friends, and his love for them. It was always stimulating to hear him tell of his friends, of the obstacles they had surmounted, the extraordinary training they had obtained, the peculiar talents they had acquired, and the

NEWS ITEMS

SOUTHERN MEDICAL ASSOCIATION

Section on Radiology

At the last meeting of the Section on Radiology of the Southern Medical Association, the following new officers were elected: *Chairman*, Charles M. Nice, Jr., M.D., Ph.D., New Orleans, Louisiana; *Vice-Chairman*, David S. Carroll, M.D., Memphis, Tennessee; and *Secretary*, Andrew F. Giesen, Jr., M.D., Fort Walton Beach, Florida.

The next meeting of the Section on Radiology will be held during the convention of the Southern Medical Association, November 1-4, 1965 in Houston, Texas.

THIRD INTERNATIONAL CONGRESS OF RADIATION RESEARCH

The Third International Congress of Radiation Research will be held in Cortina d'Ampezzo (prov. Belluno), Italy, June 26-July 2, 1966. The President of this congress is Dr. A. A. Buzzati-Traverso, International Laboratory of Genetics and Biophysics, Naples, Italy. The Secretary-General is Dr. Giovanni Silini, Laboratorio di Radiobiologie Animale, Comitato Nazionale per l'Energia Nucleare, Rome, Italy.

All inquiries about this congress should be addressed to: Secretary-General, Third International Congress of Radiation Research, Casella Postale 2359, Rome A-D, Italy.

SOUTHERN RADIOLOGICAL CONFERENCE

At the Ninth Annual Meeting of the Southern Radiological Conference, held at the Grand Hotel, Point Clear, Alabama, on January 29, 30 and 31, 1965, the following new officers were elected: Dr. Seymour F. Ochsner, *Chairman*, New Orleans, Louisiana; Dr. Everette Hoyle Schultz, *Vice-Chairman*, Chapel Hill, North Carolina; and Dr. Marshall Eskridge, *Secretary-Treasurer*, Mobile, Alabama.

The Tenth Annual Meeting will be held

at the Grand Hotel, Point Clear, Alabama, January 28, 29 and 30, 1966.

UNIVERSITY OF CINCINNATI MEDICAL CENTER

The Seventh Annual Refresher Course in Diagnostic Roentgenology will be held by the Department of Radiology of the University of Cincinnati College of Medicine under the direction of Dr. Benjamin Felson from May 24 through 28, 1965.

Further information concerning the course, which is open to Radiologists and Radiology residents, may be obtained by writing Dr. Jerome F. Wiot, Department of Radiology, Cincinnati General Hospital, Cincinnati, Ohio 45229.

MEDICAL DIAGNOSTIC APPLICATIONS OF ULTRASOUND

A conference on Medical Applications of Ultrasound will be held May 20 and 21, 1965, at the Hilton Hotel, Pittsburgh, Pennsylvania, sponsored by the Postgraduate Medical Program of the University of Pittsburgh School of Medicine.

At this conference investigators from the United States and abroad will present their experiences in the use of ultrasonic techniques for diagnostic purposes in all the various specialty areas of medicine. This will include discussion of the diagnostic applications of ultrasound in neurology and neurosurgery with particular reference to the current status of echoencephalography. It will include ultrasonic uses in cardiology and a discussion of the echocardiograph. The applications of ultrasound in ophthalmology, obstetrics and gynecology, the diagnostic application to lesions of the liver, spleen, kidney, stomach, and bladder and the use of these techniques at surgery will be discussed.

Information on registration and specific details regarding the conference may be obtained by writing to: Campbell Moses, M.D., Director, Postgraduate Medicine, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania 15213.

BOOK REVIEW

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

POSITIONING IN RADIOGRAPHY. Eighth edition. By K. C. Clark, M.B.E. Cloth. Pp. 806, with 2,539 illustrations. Price, \$35.00. Intercontinental Medical Book Corporation, 381 Park Avenue, South, New York 16, N. Y., 1964.

This widely acclaimed manual by the British radiologist, K. C. Clark, has been a familiar reference text in radiologic departments for the past quarter century. There has been no revision made since 1957, and this eighth edition fully covers the tremendous forward strides made in the specialty since that time. It is unmatched in its scope for completeness, conciseness, clarity and value to the practicing radiologist.

This edition has been enlarged by over 150 pages and almost 400 illustrations. While its principal theme is to demonstrate every conceivable positioning procedure and the results obtained therefrom, the text also encompasses the fields of radiologic anatomy, precise technical factors for proper film exposure, and typical pathologic conditions detected during routine and special procedures. It very adequately deals with such varied subjects as dental and oral radiography, ultrasound investigation, stereotaxis, gamma-ray scanning, kymography, mammography, miniature and macroradiography, lymphangiography, cineradiography and tomography, to name a few. All special procedures in each organ system are fully represented and profusely illustrated. An excellent supplemental section deals with contrast and opaque media, radiation effects and protection, and working metric equivalents.

The 2,539 illustrations have been so well selected that this manual could be termed a teaching bible for x-ray technicians. It does not enter into any but brief discussions on electrical equipment, although it does illustrate such specialized x-ray apparatus as an image intensifier. Dr. Clark holds that a good x-ray technician must be thoroughly grounded in anatomy, have some basic knowledge of physiology, and be familiar with pathologic entities which may cause him to modify his technique.

The radiographic reproductions are of a superb quality. Many of the photographs are modified with white lines to show the direction of the central and peripheral rays. In each section completely labeled film reproductions indicate anatomic structures in various projections. The terminology conforms to the *Nomina Anatomica*, Paris 1955, included in the Centenary edition of Gray's Anatomy published in 1958. All information furnished is perfectly adaptable to American radiologic practice except for minor details such as film size which follows the European pattern.

This text can truly be a comfort to the radiologist who is frequently faced with perplexing diagnostic situations. It should also be an inspiration to residents and technicians in raising the standards of radiology by obtaining the most useful information from the tools of their profession.

ARCH H. HALL, M.D.

BOOKS RECEIVED

RADIOLOGY FOR MEDICAL STUDENTS. By Fred Jenner Hodges, M.D., Professor and Chairman, Department of Radiology, University of Michigan; Isadore Lampe, M.D., Professor, Department of Radiology, University of Michigan; and John Floyd Holt, M.D., Professor, Department of Radiology, University of Michigan. Fourth edition. Cloth. Pp. 453, with 511 illustrations. Price, \$10.00. Year Book Medical Publishers, Inc., 35 East Wacker Drive, Chicago, Ill., 1965.

INTERNATIONAL SYMPOSIUM ON END RESULTS OF CANCER THERAPY. Sandefjord, Norway, September 16-20, 1963. National Cancer Institute Monograph 15. Edited by Sidney J. Cutler, National Cancer Institute, U. S. Public Health Service. Cloth. Pp. 446, with many tables. Price, \$3.75. Superintendent of Documents, U. S. Government Printing Office, Washington, D. C., 1964.

ANEURYSMS AND ARTERIOVENOUS ANOMALIES OF THE BRAIN: DIAGNOSIS AND TREATMENT. By J. Lawrence Pool, M.D., Professor of Neurological Surgery, Columbia University, College of Physicians and Surgeons; Director, Service of Neurological Surgery, Neurological Institute of New York, Columbia-Presbyterian Medical Center, New York; and D. Gordon Potts, M.D., Assistant

- Professor of Radiology, Columbia University College of Physicians and Surgeons; Assistant Attending Radiologist, Service of Radiology, Neurological Institute of New York, Columbia-Presbyterian Medical Center, New York. Cloth. Pp. 463, with many illustrations. Price, \$19.50. Hoeber Medical Division, Harper & Row, Publishers, 49 East 33rd Street, New York, N. Y., 1965.
- LA VIA LINFATICA PELVICA NELLA ONCOLOGIA GINECOLOGICA. By M. Maneschi, and P. Ragonese, with a preface by Prof. F. Marchesi, Direttore della Clinica Ostetrica e Ginecologica dell'Università di Palermo. Cloth. Pp. 321, with 145 figures. Price, L. 8,500. Luigi Pellino, Editore, Via G. B. Morgagni, 1, Rome, Italy, 1965.
- THE YEAR BOOK OF RADIOLOGY (1964-1965 Year Book Series). Radiologic Diagnosis: Edited by John Floyd Holt, M.D., Professor, Department of Radiology, University of Michigan; and Walter M. Whitehouse, M.D., Associate Professor, Department of Radiology, University of Michigan. Radiation Therapy: Edited by Howard B. Latourette, M.D., Professor, Department of Radiology, State University of Iowa. Cloth. Pp. 454, with 325 illustrations. Price, \$12.00. Year Book Medical Publishers, Inc., 35 East Wacker Drive, Chicago, Ill., 1965.
- ASSESSMENT OF RADIOACTIVITY IN MAN. Proceedings of the Symposium on the Assessment of Radioactive Body Burdens in Man, held by the International Atomic Energy Agency, International Labour Organisation and World Health Organization at Heidelberg, May 11-16, 1964. In two volumes. Cloth. Pp., First volume: 395, Second volume: 650, with a total number of 329 illustrations. Price, First volume \$8.00, Second volume, \$13.00. National Agency for International Publications, Inc., 317 East 34th Street, New York, N. Y., 1965.
- TECHNIQUE OF INTERNAL FIXATION OF FRACTURES. By M. E. Müller, M. Allgöwer, and H. Willenegger. Revised for the English edition by G. Segmüller. Cloth. Pp. 272, with 244 figures. Springer-Verlag, Berlin, 1965.



SOCIETY PROCEEDINGS

MEETINGS OF RADIOLOGICAL SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY

Secretary, Dr. C. Allen Good, Mayo Clinic, Rochester, Minn. Annual meeting: Washington Hilton Hotel, Washington, D. C., Sept. 28-Oct. 1, 1965.

AMERICAN RADIUM SOCIETY

Secretary, Dr. John L. Pool, 444 East 58th Street, New York, N. Y. 10021. Annual meeting: Camelback Inn, Phoenix, Ariz., April 13-16, 1966. (Golden Anniversary.)

RADIOLOGICAL SOCIETY OF NORTH AMERICA

Secretary-Treasurer, Dr. Maurice Doyle Frazer, 1744 South Fifty-eighth St., Lincoln, Neb. Annual meeting: Palmer House, Chicago, Ill., Nov. 28-Dec. 3, 1965.

AMERICAN COLLEGE OF RADIOLOGY

Executive Director, William C. Stronach, 20 N. Wacker Drive, Chicago 6, Ill. Annual meeting: Drake Hotel, Chicago, Ill., Feb. 1-5, 1966.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION

Secretary, Dr. Clyde A. Stevenson, Sacred Heart Hospital, West 101 Eight Ave., Spokane 4, Wash. Annual meeting: New York, N. Y., June 20-24, 1965.

AMERICAN BOARD OF RADIOLOGY

Secretary, Dr. H. Dabney Kerr. Correspondence should be directed to Kahler Hotel Building, Rochester, Minn.

The Spring 1965 examination will be held at the San Francisco Hilton Hotel, San Francisco, Calif., June 13-18, inclusive. This will be the last time that the Special Examination in Nuclear Medicine will be offered to physicians who are already certified in Radiology or Therapeutic Radiology. The deadline for filing applications for this examination was December 31, 1964.

The Fall 1965 examination will be held at the Statler Hilton Hotel, Dallas, Texas, December 5-10, inclusive. The deadline for filing applications for this examination is June 30, 1965.

AMERICAN ASSOCIATION OF PHYSICISTS IN MEDICINE

Secretary-Treasurer, Charles S. Simons, University of Michigan Hospital, Ann Arbor, Mich. Annual meeting to be announced.

AMERICAN CLUB OF THERAPEUTIC RADIOLOGISTS

Secretary, Dr. J. A. del Regato, Penrose Cancer Hospital, Colorado Springs, Colo.

ELEVENTH INTERNATIONAL CONGRESS OF RADIOLOGY

Secretary-General, Professor Dr. Med. Arduino Ratti, via Moscovia, 44-1, Milan, Italy. Address inquiries to Professor Dr. Med. Luigi Turano, President-Elect, Istituto di Radiologia, Università di Roma, Rome, Italy. Meeting: September 22-28, 1965.

NINTH INTER-AMERICAN CONGRESS OF RADIOLOGY

Counselor for the United States, Dr. Philip J. Hodes, Jefferson Medical College Hospital, 11th and Walnut Streets, Philadelphia 7, Pennsylvania. Meeting: Montevideo, Uruguay, 1967.

ALABAMA RADIOLOGICAL SOCIETY

Secretary, Dr. Walter Brower, Birmingham, Ala. Meets time and place of Alabama State Medical Association.

AMERICAN NUCLEAR SOCIETY

Treasurer, Raymond Maxson, 86 E. Randolph St., Chicago, Ill. Annual meeting to be announced.

ARIZONA RADIOLOGICAL SOCIETY

Secretary-Treasurer, Dr. George Gentner, 3435 W. Durango, Phoenix, Ariz. Two regular meetings a year. Annual meeting at time and place of State Medical Association and interim meeting six months later.

ARKANSAS RADIOLOGICAL SOCIETY

Secretary, Dr. Charles W. Anderson, 1108½ Poplar, Pine

Bluff, Ark. Meets every three months and also at time and place of State Medical Association.

ASSOCIATION OF UNIVERSITY RADIOLOGISTS

Secretary-Treasurer, Dr. Alexander R. Margulis, Department of Radiology, University of California, San Francisco, Calif. Annual meeting: Seattle, Washington, May 14 and 15, 1965.

ATLANTA RADIOLOGICAL SOCIETY

Secretary, Dr. Donald R. Rooney, Burnt Hickory Road, Marietta, Ga. Meets monthly except during three summer months, on third Tuesday, at the Academy of Medicine, Atlanta, Ga., at 8:00 P.M.

BAVARIAN-AMERICAN RADIOLOGICAL SOCIETY

Secretary, Dr. Roy R. Deffebach, Major, MC, Radiology Service, 5th General Hospital, APO 154, New York, N. Y. Meets quarterly.

BLOCKLEY RADIOLOGICAL SOCIETY

Secretary, Dr. Bernard J. Ostrum, 2412 North 52nd St., Philadelphia, Pa.

BLUEGRASS RADIOLOGICAL SOCIETY

Secretary-Treasurer, Dr. Arthur Lieber, University of Kentucky, University Hospital, Lexington, Kentucky. Meets quarterly.

BROOKLYN RADIOLOGICAL SOCIETY

Secretary, Dr. Jay P. Sackler, Brooklyn, N. Y. Meets first Thursday of each month, October through June.

BUFFALO RADIOLOGICAL SOCIETY

Secretary, Dr. Berkeley Zinn, 85 Wehrle Dr., Buffalo 25, N. Y. Meets second Monday evening each month, October to May inclusive.

CALIFORNIA RADIOLOGICAL SOCIETY

Secretary, Dr. L. Henry Garland, Suite 1739, 450 Sutter St., San Francisco, Calif. Meets annually during meeting of California Medical Association.

CATAWBA VALLEY RADIOLOGICAL SOCIETY

Secretary, Dr. Emmett R. White, P. O. Box 303, Rutherford College, N. C. Meets every Tuesday, Dept. of Radiology, Valde General Hosp., Valde, N. C. at 12:00 P.M.

CENTRAL NEW YORK RADIOLOGICAL SOCIETY

Secretary-Treasurer, Dr. Edward W. Carsky, Crouse-Irving Hospital, 820 S. Crouse Ave., Syracuse, N. Y. Meets first Monday each month, October through May.

CENTRAL OHIO RADIOLOGICAL SOCIETY

Secretary-Treasurer, Dr. Harold W. Long, 81 S. Fifth St., Columbus 15, Ohio. Meets third Thursday in October, and second Thursday in November, January, March and May at Fort Hayes Hotel, Columbus, Ohio.

CENTRAL SOCIETY OF NUCLEAR MEDICINE

Secretary, Dr. Robert S. Landauer, Radiation Center Bldg., 1903 West Harrison St., Chicago 12, Ill.

CHICAGO ROENTGEN SOCIETY

Secretary-Treasurer, Dr. Robert D. Moseley, Jr., Dept. of Radiology, Univ. of Chicago, 950 E. 59th St., Chicago 37, Ill. Meets second Thursday of each month, October to April except December at the Pick-Congress Hotel at 8:00 P.M.

CLEVELAND RADIOLOGICAL SOCIETY

Secretary-Treasurer, Dr. James Christie, 10515 Carnegie Avenue, Cleveland, Ohio. Meetings at 7:00 P.M. on fourth Monday of October, November, January, February, March and April.

COLORADO RADIOLOGICAL SOCIETY

Secretary, Dr. David J. Stephenson, 8300 West 38th Ave., Wheat Ridge, Colo. Meets third Friday of each month at Denver Athletic Club from September through May.

* Secretaries of societies are requested to send timely information promptly to the Editor.

CONNECTICUT VALLEY RADIOLOGIC SOCIETY

Secretary, Dr. William W. Walthall, Jr., 130 Maple St., Springfield, Mass. Meets in April and October.

DALLAS-FORT WORTH RADIOLOGICAL SOCIETY

Secretary, Dr. R. E. Collier, 3500 Gaston Ave., Dallas, Tex. Meets monthly, third Monday, at Southwest International Airport at 6:30 P.M.

DETROIT ROENTGEN RAY AND RADIUM SOCIETY

Secretary, Dr. Arch H. Hall, Harper Hospital, Detroit 1, Mich. Meets monthly, first Thursday, October through May, at David Whitney House, 1010 Antietam, at 6:30 P.M.

EAST BAY ROENTGEN SOCIETY

Secretary, Dr. William G. Faraghan, 450 30th St., Oakland 9, Calif. Meets first Thursday each month at University Club, Oakland, Calif.

EAST TENNESSEE RADIOLOGICAL SOCIETY

Secretary, Dr. C. H. Kimball, 2200 Harris Circle, Cleveland, Tenn. Meets in January and September.

EASTERN RADIOLOGICAL SOCIETY

Secretary, Dr. James F. Martin, North Carolina Baptist Hospital, Winston-Salem, N. C.

FLORIDA RADIOLOGICAL SOCIETY

Secretary, Dr. Andre S. Capi, 3471 N. Federal Highway, Fort Lauderdale, Fla. Meets twice annually, in the spring with the annual State Society Meeting and in the fall.

FLORIDA WEST COAST RADIOLOGICAL SOCIETY

Secretary-Treasurer, Dr. Garth R. Drewry, Tampa General Hospital, Tampa 6, Fla. Meets in January, April, July and October.

GEORGIA RADIOLOGICAL SOCIETY

Secretary, Dr. David Robinson, P.O. Box 394, Savannah, Ga. Meets in spring and fall with Annual State Society Meeting.

GREATER MIAMI RADIOLOGICAL SOCIETY

Secretary-Treasurer, Dr. Oliver P. Winslow, Jr., Baptist Hospital of Miami, Inc., 8900 S. W. 88th St., Miami 56, Fla. Meets monthly, third Wednesday at 8:00 P.M. at Jackson Memorial Hospital, Miami, Fla.

GREATER ST. LOUIS SOCIETY OF RADIOLOGISTS

Secretary-Treasurer, Dr. Mark D. Eagleton, 950 Francis Place, St. Louis, Mo.

HOUSTON RADIOLOGICAL SOCIETY

Secretary, Dr. D. A. Van Velzer, Texas Medical Center Library, Jesse H. Jones Library Bldg., Houston 25, Tex. Meets fourth Monday of each month, except June, July, August and December, at the Doctors' Club, 8:00 P.M., Houston, Tex.

IDAHO STATE RADIOLOGICAL SOCIETY

Secretary, Dr. George H. Harris, Bannock Memorial Hospital, Pocatello, Idaho. Meets in the spring and fall.

ILLINOIS RADIOLOGICAL SOCIETY

Secretary, Dr. George A. Miller, Carle Hospital Clinic, Urbana, Ill. Meets in the spring and fall.

INDIANA ROENTGEN SOCIETY, INC.

Secretary, Dr. Richard A. Silver, 1815 N. Capitol Avenue, Indianapolis, Ind. Meets first Sunday in May and during fall meeting of Indiana State Medical Association.

IOWA RADIOLOGICAL SOCIETY

Secretary, Dr. L. L. Maher, 1419 Woodland Ave., Des Moines, Iowa. Luncheon and business meeting during annual session of Iowa State Medical Society. The scientific section is held in the autumn.

KANSAS RADIOLOGICAL SOCIETY

Secretary-Treasurer, Dr. Robert C. Lawson, 310 Medical Arts Bldg., 10th and Home, Topeka, Kan. Meets in spring with State Medical Society and in winter on call.

KENTUCKY CHAPTER, AMERICAN COLLEGE OF RADIOLOGY

Secretary-Treasurer, Dr. Robert H. Greenlaw, Dept. of Radiology, Univ. of Kentucky Med. Ctr., Lexington, Ky. Meetings will be semiannually.

KENTUCKY RADIOLOGICAL SOCIETY

Secretary-Treasurer, Dr. Joan R. Hale, 402 Heyburn Building, Louisville, Ky. Meets monthly on second Friday at Sheraton Hotel, Louisville, Ky.

KINGS COUNTY RADIOLOGICAL SOCIETY

Secretary, Dr. Stanley Dannenberg, 1917 Bedford Ave., Brooklyn 25, N. Y. Meets Kings County Med. Soc.

Bldg. monthly on fourth Thursday, October to May, 8:45 P.M.

KNOXVILLE RADIOLOGICAL SOCIETY

Secretary, Dr. Clifford L. Walton, Blount Professional Bldg., Knoxville 20, Tenn. Meetings are held the third Monday of every other month at the University of Tennessee Memorial Research Center and Hospital.

LONG ISLAND RADIOLOGICAL SOCIETY

Secretary, Dr. Perry R. Mandel, Nassau Hospital, Mineola, L. I., N. Y. Meets second Tuesday of the month in February, April, June, October and December.

LOS ANGELES RADIOLOGICAL SOCIETY

Secretary, Dr. Austin R. Wilson, 540 N. Central Ave., Glendale, Calif. 91203. Meets second Wednesday of month in November, January, April and June at Los Angeles County Medical Association Building, Los Angeles, Calif.

MAINE RADIOLOGICAL SOCIETY

Secretary, Dr. J. T. Chen, 7 Cherry Hill Terrace, Waterville, Me. Meets in June, September, December and April.

MARYLAND RADIOLOGICAL SOCIETY

Secretary, Dr. Henry Startzman, Medical Arts Building, Baltimore, Maryland.

MEMPHIS ROENTGEN SOCIETY

Secretary-Treasurer, Dr. Irving K. Ettman, 684 W. Brookhaven Circle, Memphis, Tenn. Meets first Monday of each month at John Gaston Hospital.

MIAMI VALLEY RADIOLOGICAL SOCIETY

Secretary, Dr. William D. Roberts, 2197 Los Arrow Dr., Dayton 9, Ohio. Meets second Friday of fall and winter months.

MID-HUDSON RADIOLOGICAL SOCIETY

Secretary-Treasurer, Dr. Alexander W. Friedman, Mid-Hudson Medical Group, Fishkill, N. Y. Meets 7:00 P.M., first Wednesday of each month September to May.

MILWAUKEE ROENTGEN RAY SOCIETY

Secretary-Treasurer, Dr. Harold F. Ibach, 2400 W. Vilard Avenue, Milwaukee 9, Wis. Meets monthly on fourth Monday, October through May, at University Club.

MINNESOTA RADIOLOGICAL SOCIETY

Secretary-Treasurer, Dr. Frank J. Anderson, 810 E. 27th St., Minneapolis 7, Minn. Meets three times annually, in fall, winter and spring.

MISSISSIPPI RADIOLOGICAL SOCIETY

Secretary-Treasurer, Dr. Dan T. Keel, Jr., 504 Chippewa St., Brookhaven, Miss. Meets third Thursday of each month at the Heidelberg Hotel, Jackson, at 6:00 P.M.

MISSOURI RADIOLOGICAL SOCIETY

Secretary-Treasurer, Dr. M. Shoss, Cape Girardeau, Mo.

MONTANA RADIOLOGICAL SOCIETY

Secretary, Dr. Clark Grimm, Great Falls, Montana. Meets at least once a year.

NEBRASKA STATE RADIOLOGICAL SOCIETY

Secretary, Dr. Richard Bunting, The Radiologic Center, Nebraska Methodist Hospital, Omaha 31, Neb. Meets third Wednesday of each month at 6 P.M. in Omaha or Lincoln.

NEVADA RADIOLOGICAL SOCIETY

Secretary, Dr. William G. Arbonies, Department of Radiology, St. Mary's Hospital, Reno, Nev.

NEW ENGLAND ROENTGEN RAY SOCIETY

Secretary, Dr. Jack R. Dreyfuss, Zero Emerson Place, Boston, Mass. 02114. Meets third Friday of each month, October through April, at The Longwood Towers, 20 Chapel Street, Brookline, Mass., at 4:30 P.M.

NEW HAMPSHIRE ROENTGEN RAY SOCIETY

Secretary, Dr. Paul Y. Hassserjian, 1470 Elm St., Manchester, N. H. Meets four to six times yearly.

NEW MEXICO ASSOCIATION OF RADIOLOGISTS

Secretary-Treasurer, Dr. Justin J. Wolfson, Department of Radiology, Bernalillo County-Indian Hospital, Albuquerque, New Mexico.

NEW MEXICO SOCIETY OF RADIOLOGISTS

Secretary, Dr. William G. McPherson, Hobbs, New Mexico. Four annual meetings, three held in Albuquerque.

- que, N. M., and one held at time and place of New Mexico State Medical Society annual meeting.
- NEW YORK ROENTGEN SOCIETY**
Secretary, Dr. Harry Z. Mellins. Meets monthly on third Monday at the New York Academy of Medicine at 4:30 P.M.
- NORTH CAROLINA RADIOLOGICAL SOCIETY**
Secretary, Dr. E. H. Schultz, North Carolina Memorial Hospital, Chapel Hill, N. C. Meets in the spring and fall each year.
- NORTH DAKOTA RADIOLOGICAL SOCIETY**
Secretary, Dr. John Jestadt, Depuy-Sorkness Clinic, Jamestown, N. D. Meets at time of State Medical Association meeting. Other meetings arranged on call of the President.
- NORTH FLORIDA RADIOLOGICAL SOCIETY**
Secretary, Dr. Charles H. Newell, 800 Miami Road, Jacksonville 7, Fla. Meets quarterly in March, June, September and December.
- NORTHEASTERN NEW YORK RADIOLOGICAL SOCIETY**
Secretary, Dr. Anthony J. Tabacco, 621 Central Ave., Albany 6, N. Y. Meets in Albany area on second Wednesday of October, November, March and April.
- NORTHERN CALIFORNIA RADIOLOGICAL SOCIETY**
Secretary, Dr. John M. Johannessen, Mercy Hospital, Sacramento, Calif. Meets fourth Monday of Sept., Nov., Jan., March and May at the Sutter Club in Sacramento.
- NORTHWESTERN OHIO RADIOLOGICAL SOCIETY**
Secretary, Dr. Vito J. Zupa, Mercy Hospital, Department of Radiology, Toledo, Ohio.
- OHIO STATE RADIOLOGICAL SOCIETY**
Secretary, Dr. M. M. Thompson, Jr., 316 Michigan St., Toledo 2, Ohio. Annual meeting: Terrace Hilton Hotel, Cincinnati, Ohio, May, 1965.
- OKLAHOMA STATE RADIOLOGICAL SOCIETY**
Secretary, Dr. Dave B. Lhevine, Bio Doctors Bldg., 2021 South Lewis, Tulsa, Okla. Meets in January, May and October.
- ORANGE COUNTY RADIOLOGICAL SOCIETY**
Secretary-Treasurer, Dr. Herbert H. Benson, 100 East Valencia-Mesa Dr., Fullerton, Calif. Meets fourth Tuesday of every month in Orange County Medical Association Building.
- OREGON RADIOLOGICAL SOCIETY**
Secretary-Treasurer, Dr. James A. Schneider, St. Vincent Hospital, Portland 10, Ore. Meets on second Wednesday of month at 7:00 P.M. at the University Club, Portland, Ore.
- ORLEANS PARISH RADIOLOGICAL SOCIETY**
Secretary, Dr. Joseph V. Schlosser, Charity Hospital, New Orleans 13, La. Meets second Tuesday of each month.
- PACIFIC NORTHWEST RADIOLOGICAL SOCIETY**
Secretary, Dr. Willis Taylor, Seattle, Washington. Annual meeting: Olympic Hotel, Seattle, Washington, May 7-9, 1965.
- PENNSYLVANIA RADIOLOGICAL SOCIETY**
Secretary-Treasurer, Dr. Frederick R. Gilmore, Clearfield Hospital, Clearfield, Pa. Annual meeting: Pittsburgh Hilton Hotel, May 21-22, 1965.
- PHILADELPHIA ROENTGEN RAY SOCIETY**
Secretary, Dr. Antolin Raventos, Hospital of the University of Pennsylvania, Philadelphia 4, Pa. Meets first Thursday of each month at 5 P.M., from October to May in Thompson Hall, College of Physicians.
- PITTSBURGH ROENTGEN SOCIETY**
Secretary, Dr. Robert N. Berk, 3305 Fifth Ave., Pittsburgh 13, Pa. Meets second Wednesday of month, October through June at Park Schenely Restaurant.
- RADIOLOGICAL SOCIETY OF CONNECTICUT**
Secretary-Treasurer, Dr. Orlando F. Gabriele, 1450 Chapel St., New Haven 11, Conn. Meetings are held bimonthly.
- RADIOLOGICAL SOCIETY OF GREATER CINCINNATI**
Secretary, Dr. Harold N. Margolin, 6159 Tulane Road, Cincinnati, Ohio. Meets first Monday of each month at Cincinnati Academy of Medicine.
- RADIOLOGICAL SOCIETY OF GREATER KANSAS CITY**
Secretary, Dr. J. Stewart Whitmore, 1010 Rialto Bldg., Kansas City, Mo. Meets last Friday of each month.
- RADIOLOGICAL SOCIETY OF HAWAII**
Secretary, Dr. Russell E. Graf, P.O. Box 294, USA Tripler General Hospital, Honolulu, Hawaii. Meets third Monday of each month at 7:30 P.M.
- RADIOLOGICAL SOCIETY OF KANSAS CITY**
Secretary, Dr. Arthur B. Smith, 800 Argyle Bldg., Kansas City, Mo. Meets third Thursday of each month.
- RADIOLOGICAL SOCIETY OF LOUISIANA**
Secretary, Dr. Andrew F. Giesen, Ochsner Clinic, New Orleans 15, La. Meets annually during Louisiana State Medical Society meeting.
- RADIOLOGICAL SOCIETY OF NEW JERSEY**
Secretary, Dr. E. Arthur Kratzman, 912 Prospect Ave., Plainfield, N. J. Meets in Atlantic City at time of State Medical Society meeting and in October or November in Newark, N. J.
- RADIOLOGICAL SOCIETY OF RHODE ISLAND**
Secretary-Treasurer, Dr. Harvey B. Lesselbaum, Miriam Hospital, Providence, R. I.
- RADIOLOGICAL SOCIETY OF THE STATE OF NEW YORK**
Secretary-Treasurer, Dr. John W. Colgan, 273 Hollywood Ave., Rochester 18, N. Y.
- RADIOLOGICAL SOCIETY OF SOUTH DAKOTA**
Secretary-Treasurer, Dr. Donald J. Peik, 303 S. Minnesota Ave., Sioux Falls, S. D.
- RADIOLOGICAL SOCIETY OF SOUTHERN CALIFORNIA**
Secretary-Treasurer, Dr. John L. Gwinn, Children's Hospital of Los Angeles, 4614 Sunset Blvd., Los Angeles, Calif. 90027. Meets three times a year, usually October, February, and May.
- REDWOOD EMPIRE RADIOLOGICAL SOCIETY**
Secretary, Dr. Lee F. Titus, 164 W. Napa St., Sonoma, Calif. Meets second Monday every other month.
- RICHMOND COUNTY RADIOLOGICAL SOCIETY**
Secretary, Dr. W. F. Hamilton, Jr., University Hospital, Augusta, Ga. Meets first Thursday of each month at various hospitals.
- ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.**
Secretary, Dr. Irving B. Joffe, Rochester General Hospital, 1425 Portland Ave., Rochester 21, N. Y. Meets at 8:15 P.M. on the last Monday of each month, September through May, at Strong Memorial Hospital.
- ROCKY MOUNTAIN RADIOLOGICAL SOCIETY**
Secretary, Dr. John H. Freed, 4200 East Ninth Ave., Denver 20, Colo. Annual meeting to be announced.
- SAN ANTONIO-MILITARY RADIOLOGICAL SOCIETY**
Secretary, Dr. Hugh F. Elmendorf, Jr., 730 Medical Arts Bldg., San Antonio 5, Tex. Meets third Wednesday each month in Fort Sam Houston Officer's Club at 6:30 P.M.
- SAN DIEGO RADIOLOGICAL SOCIETY**
President-Secretary, Charles P. Hyslop, 7901 Frost St., San Diego 22, Calif. Meets first Wednesday of each month at the University Club.
- SAN FRANCISCO RADIOLOGICAL SOCIETY**
Secretary, Dr. Malcolm Jones, University of California Hospital, San Francisco 22, Calif. Meets quarterly at the San Francisco Medical Society, 250 Masonic Ave., San Francisco 18, Calif.
- SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION**
Secretary, Dr. William H. Graham, 630 East Santa Clara St., San Jose, Calif.
- SECTION ON RADIOLOGY, MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA**
Secretary-Treasurer, Dr. Martin A. Thomas, 1150 Connecticut Ave., Washington 36, D. C. Meets at Medical Society Library, third Wednesday of January, March, May and October at 8:00 P.M.
- SECTION ON RADIOLOGY, SOUTHERN MEDICAL ASSOCIATION**
Secretary, Dr. Andrew F. Giesen, Jr., White-Wilson Clinic, Fort Walton Beach, Fla. Annual meeting: Houston, Texas, Nov. 1-4, 1965.
- SECTION ON RADIOLOGY, TEXAS MEDICAL ASSOCIATION**
Secretary, Dr. George F. Crawford, St. Elizabeth Hospi-

- tal, Beaumont, Tex. Meets annually with the Texas Medical Association.
- SHREVEPORT RADIOLOGICAL CLUB**
Secretary, W. R. Harwell, 608 Travis St., Shreveport, La. Meets monthly on third Wednesday at 7:30 P.M., September to May inclusive.
- SOCIETY FOR PEDIATRIC RADIOLOGY**
Secretary, Dr. John L. Gwinn, Children's Hospital, Los Angeles 27, Calif. Annual meeting: Washington Hilton Hotel, Washington, D. C., September 27, 1965.
- SOCIETY OF NUCLEAR MEDICINE**
Secretary, Mr. C. Craig Harris, Oak Ridge National Laboratories, Oak Ridge, Tenn. *Administrator*, Mr. Samuel N. Turiel, 430 N. Michigan Ave., Chicago 11, Ill. Annual meeting: Bal Harbour, Fla., June 17-19, 1965.
- SOUTH BAY RADIOLOGICAL SOCIETY**
Secretary, Northern Section: Dr. John H. Callaghan, 2900 Whipple Ave., Redwood City, Calif.; Southern Section: Dr. Paul L. Clemetson, 877 W. Fremont Ave., Suite C-3, Sunnyvale, Calif. Meets second Wednesday of each month.
- SOUTH CAROLINA RADIOLOGICAL SOCIETY**
Secretary, Dr. George W. Brunson, 1406 Gregg St., Columbia, S. C. Annual meeting (primarily business) in conjunction with the South Carolina Medical Association meeting in May. Annual fall scientific meeting at time and place designated by the president.
- SOUTH DAKOTA RADIOLOGICAL SOCIETY**
Secretary, Dr. Donald J. Peik, 1417 S. Minnesota Ave., Sioux Falls, S. Dak. Meets in spring with State Medical Society and in fall.
- SOUTHERN RADIOLOGICAL CONFERENCE**
Secretary *Treasurer*, Dr. Marshall Eskridge, Mobile Infirmary, P.O. Box 4097, Mobile, Ala. Annual meeting: Grand Hotel, Point Clear, Ala., Jan. 28-30, 1966.
- SOUTHWESTERN RADIOLOGICAL SOCIETY**
Secretary, John M. McGuire, 904 Chelsea, El Paso, Tex. Meets last Monday of each month at 6:30 P.M. in the Paso del Norte Hotel.
- TENNESSEE RADIOLOGICAL SOCIETY**
Secretary, Dr. B. M. Brady, St. Joseph Hospital, Memphis, Tenn. Meets annually at the time and place of the Tennessee State Medical Association meeting.
- TEXAS RADIOLOGICAL SOCIETY**
Secretary, Dr. Herman C. Sehested, 815 Medical Arts Bldg., Fort Worth 2, Tex. Annual meeting: Driscoll Hotel, Corpus Christi, Jan. 28-29, 1966.
- TRI-STATE RADIOLOGICAL SOCIETY**
Secretary, Dr. John H. Marchand, Jr., Methodist Hospital, Henderson, Ky. Meets third Wednesday of Oct., Jan., March and May, 8:00 P.M., Elks Club in Evansville, Ind.
- UNIVERSITY OF MICHIGAN DEPARTMENT OF ROENTGENOLOGY STAFF MEETING**
 Meets each Monday evening from September to June, at 7:00 P.M. at University Hospital, Ann Arbor, Mich.
- UPPER PENINSULA RADIOLOGICAL SOCIETY**
Secretary, Dr. A. Gonty, Menominee, Mich. Meets quarterly.
- UTAH STATE RADIOLOGICAL SOCIETY**
Secretary, Dr. Carlisle C. Smith, Salt Lake General Hospital, 2033 S. State St., Salt Lake City, Utah. Meets fourth Wednesday in January, March, May, September and November at Holy Cross Hospital.
- VERMONT RADIOLOGICAL SOCIETY**
Secretary, Dr. John R. Williams, 160 Allen St., Rutland, Vt.
- VIRGINIA RADIOLOGICAL SOCIETY**
Secretary, Dr. John M. Ratliff, Mary Immaculate Hospital, Newport News, Va.
- WASHINGTON STATE RADIOLOGICAL SOCIETY**
Secretary, Dr. Owen Marten, 930 Terry Avenue, Seattle, Wash. Meets quarterly.
- WEST VIRGINIA RADIOLOGICAL SOCIETY**
Secretary, Dr. Karl J. Myers, The Myers Clinic-Broadus Hospital, Philippi, W. Va. Meets concurrently with Annual Meeting of West Virginia State Medical Society; other meetings arranged by program committee.
- WESTCHESTER RADIOLOGICAL SOCIETY**
Secretary, Dr. Joel J. Schwartzman, Westchester Academy of Medicine, Section on Radiology, Purchase, N. Y. Meets on third Tuesday of January and October and on two other dates.
- WISCONSIN RADIOLOGICAL SOCIETY**
Secretary, Dr. Charles Benkendorf, 408 St. Francis St., Green Bay, Wis. Meets twice a year, May and September.
- CUBA, MEXICO, PUERTO RICO AND CENTRAL AMERICA**
- ASOCIACIÓN DE RADIOLOGOS DE CENTRO AMERICA Y PANAMÁ.** Comprising: Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica and Panamá.
Secretary-General, Dr. Roberto Calderón, Calle Central Oeste No. 218, Managua, Nicaragua, Central America. Meets annually in a rotating manner in the six countries.
- SOCIEDAD DE RADIOLOGÍA DE EL SALVADOR**
Secretary, Dr. Rafael Vaga Gómez.
- SOCIEDAD DE RADIOLOGÍA DE GUATEMALA**
Secretary, Dr. Carlos E. Escobar, 92. Calle A-0-05, Zona 1, Guatemala.
- SOCIEDAD DE RADIOLOGÍA Y FISIOTERAPIA CUBANA**
Secretary, Dr. Miguel A. García Plasencia, Hospital Curie, 29 y F, Vedado, Habana, Cuba. Meets monthly at Curie Hospital.
- SOCIEDAD COSTARRICENSE DE RADIOLOGIA**
Secretary, Dr. James Fernández Carballo, Apartado VIII, San José, Costa Rica.
- SOCIEDAD MEXICANA DE RADIOLOGÍA, A.C.**
 Calle del Oro No. 15, México 7, D. F.
Secretary-General, Dr. E. Alvarez Hernández.
 Meets first Monday of each month.
- ASOCIACIÓN PUERTORRIQUEÑA DE RADIOLOGÍA**
Secretary, Dr. R. B. Díaz Bonnet, Suite 504, Professional Bldg., Santurce, Puerto Rico.
- SOCIEDAD RADIOLOGICA PANAMEÑA**
Secretary, Dr. L. Arrieta Sánchez, Apartado No. 6323, Panamá, R. de P. Meets monthly in a department of radiology of a local hospital chosen at preceding meeting.
- SOCIEDAD RADIOLOGICA DE PUERTO RICO**
Secretary, Dr. Jorge Carreras Girard, Suite 504, Professional Bldg., Santurce, Puerto Rico. Meets second Thursday of each month at 8:00 P.M. at the Puerto Rico Medical Association Bldg. in San Juan.
- BRITISH COMMONWEALTH OF NATIONS**
- ASSOCIATION OF RADIOLOGISTS OF THE PROVINCE OF QUEBEC**
Secretary, Dr. R. Robillard, Notre-Dame Hospital, 1560 Sherbrooke St., East, Montreal, Que., Canada. Meets four times a year.
- BRITISH INSTITUTE OF RADIOLOGY**
Honorary Secretary, Dr. R. D. Hoare, 32 Welbeck St., London, W. 1, England. Meets monthly from October until May.
- CANADIAN ASSOCIATION OF PHYSICISTS, DIVISION OF MEDICAL AND BIOLOGICAL PHYSICS.**
Honorary Secretary-Treasurer, Paul M. Pfalzner, Dept. of Therapeutic Radiology, University of Western Ontario, London, Ont., Canada. Annual meeting to be announced.
- EDMONTON AND DISTRICT RADIOLOGICAL SOCIETY**
Secretary, Dr. J. T. Mason, 540 Tegler Bldg., Edmonton, Alberta, Canada. Meets second Tuesday of each month, October to May.
- FACULTY OF RADIOLOGISTS**
Honorary Secretary, Dr. J. N. Pattinson, 47 Lincoln's Inn Fields, London, W.C.2, England. Annual meeting: Cambridge, England, June 18-19, 1965.
- FACULTY OF RADIOLOGISTS, ROYAL COLLEGE OF SURGEONS IN IRELAND**
Registrar, Dr. H. O'Flanagan, F.R.C.P.I., D.P.H., 123 St. Stephens Green, Dublin 2, Ireland.
- SECTION OF RADIOLOGY OF THE ROYAL SOCIETY OF MEDICINE (CONFINED TO MEDICAL MEMBERS)**
 Meets third Friday each month at 4:45 P.M. at the Royal

- Society of Medicine, 1 Wimpole St., London, W. 1, England.
- CANADIAN ASSOCIATION OF RADIOLOGISTS
Honorary Secretary-Treasurer, Dr. D. J. Sierewicz, *Associate Honorary Secretary-Treasurer*, Dr. Maurice Dufresne, 1555 Summerhill Ave., Montreal 25, Que., Canada. Annual meeting to be announced.
- MONTREAL RADIOLOGICAL STUDY CLUB
Secretary, Dr. Leonard Rosenthal, Montreal General Hospital, Montreal, Que., Canada. Meets first Tuesday evening, October to April.
- SECTION OF RADIOLOGY, CANADIAN MEDICAL ASSOCIATION
Secretary, Dr. C. M. Jones, Inglis St., Ext. Halifax, N. S.
- SOCIÉTÉ CANADIENNE-FRANÇAISE D'ELECTRO-RADIOLOGIE MÉDICALE
General Secretary, Dr. Maurice Dufresne, 1560 Sherbrooke (East), Montreal, Que., Canada. Meets third Saturday each month.
- TORONTO RADIOLOGICAL SOCIETY
Secretary, Dr. Wallace M. Roy, St. Joseph's Hospitals, 30 The Queensway, Toronto 3, Ont., Canada. Meets second Monday of each month, September through May.
- COLLEGE OF RADIOLOGISTS OF AUSTRALASIA
Honorary Secretary, Dr. E. A. Booth, c/o British Medical Agency, 135 Macquarie St., Sydney, N.S.W., Australia.
- SOUTH AMERICA
- ASOCIACIÓN ARGENTINA DE RADIOLOGÍA
Secretary, Dr. Lidio G. Mosca, Avda. G. al. Paz 151, Córdoba, Argentina. Meetings held monthly.
- ATENEO DE RADIOLOGÍA
Secretary, Dr. Victor A. Añños, Instituto de Radiología, Santa Fe 3100, Rosario, Argentina. Meets monthly on second and fourth Fridays at 7:00 P.M. in the Hospital Nacional de Centenario, Santa Fe 1300, Rosario.
- COLÉGIO BRASILEIRO DE RADIOLOGIA
Secretary-General, Dr. Tede Eston de Eston, Caixa Postal 5984, São Paulo, Brazil.
- SOCIEDAD ARGENTINA DE RADIOLOGIA
Secretary, Dr. Armando B. de Onaindia, Santa Fe 1171, Buenos Aires. Meetings are held monthly.
- SOCIEDAD BOLIVIANA DE RADIOLOGÍA
Secretary, Dr. Javier Prada Méndez, Casilla 1596, La Paz, Bolivia. Meets monthly. General assembly once every two years.
- SOCIEDADE BRASILEIRA DE RADIOLOGIA
Secretary, Dr. Nicola Caminha, Av. Mem. de Sa, Rio de Janeiro, Brazil. General Assembly meets every two years in December.
- SOCIEDADE BRASILEIRA DE RADIOTERAPIA
Secretary, Dr. Oscar Rocha von Pfuhl, Av. Brigadeiro Luiz Antonio, 644, São Paulo, Brazil. Meets monthly on second Wednesday at 9:00 P.M. in São Paulo at Av. Brigadeiro Luiz Antonio, 644.
- SOCIEDAD CHILENA DE RADIOLOGÍA
Secretary, Dr. J. P. Velasco, Avenida Santa María 0410, Santiago, Chile. Meets fourth Friday of each month.
- SOCIEDAD COLOMBIANA DE RADIOLOGIA
Secretary, Dr. Armando Uribe, Hospital Militar Central, Apartado aéreo No. 5804, Bogotá, Colombia. Meets last Thursday of each month.
- SOCIEDAD ECUATORIANA DE RADIOLOGÍA Y FISIOTERAPIA
Secretary, Dr. Luis Blum, P.O. Box 3711, Guayaquil, Ecuador.
- SOCIEDAD PARAGUAYA DE RADIOLOGÍA
Secretary, Dr. Miguel González Addone, 15 de Agosto 322, Asunción, Paraguay.
- SOCIEDAD PERUANA DE RADIOLOGIA
Secretary, Dr. Vicente Ubillus, Apartado 2306, Lima, Peru. Meets monthly except during January, February and March, at Asociación Médica Peruana "Daniel A. Carrión," Villalta 218, Lima.
- SOCIEDAD DE RADIOLOGICA DEL ATLANTICO
Secretary, Dr. Raul Fernandez, Calle 40 #4 -110, Baranquilla, Colombia. Society meets monthly at the Instituto de Radiologia.
- SOCIEDAD DE RADIOLOGÍA, CANCEROLOGÍA Y FÍSICA MÉDICA DEL URUGUAY
Secretary-General, Dr. Ernesto H. Cibils, Av. Agraciada 1464, piso 13, Montevideo, Uruguay.
- SOCIEDADE DE RADIOLOGIA DE PERNAMBUCO
Secretary, Dr. Manoel Medeiros, Instituto de Radiologia da Faculdade de Medicina da Universidade do Recife, Caixa Postal 505, Pernambuco, Brazil.
- SOCIEDAD DE ROENTGENOLOGIA Y MEDICINA NUCLEAR DE LA PROVINCIA DE CÓRDOBA
Secretary-General, Dr. Carlos A. Oulton, Santa Rosa 447, Córdoba, Argentina.
- SOCIEDAD VENEZOLANA DE RADIOLOGÍA
Secretary-General, Dr. Rubén Merensfeld, Apartado No. 9362 Candelaria, Caracas, Venezuela. Meets monthly third Friday at Colegio Médico del Distrito Federal, Caracas.
- CONTINENTAL EUROPE
- ÖSTERREICHISCHE RÖNTGEN-GESELLSCHAFT
President, Dr. Konrad Weiss, Mariannengasse 10, Vienna 9, Austria. Meets second Tuesday of each month in Allgemeine Poliklinik. Annual meeting to be announced.
- SOCIÉTÉ BELGE DE RADIOLOGIE
General Secretary, Prof. Simon Masy, Louvain, Belgium. Meets in February, March, May, June, September, October, November and December.
- SOCIÉTÉ EUROPÉENNE DE RADIOLOGIE PÉDIATRIQUE
Permanent Secretary, Dr. Jaques Sauvegrain, Hôpital des Enfants-Malades, 149, rue de Sèvres, Paris 15e, France. *General Secretary*, Dr. Ole Eklöf, P.O. Box, Stockholm 60, Sweden. Annual meeting: Stockholm, Sweden, May 20-22, 1965.
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 The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association.
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- INDIAN RADIOLOGICAL ASSOCIATION
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- INDONESIAN RADIOLOGICAL SOCIETY
Secretary, Professor Sjahriar Rasad, Taman Tjut Mutiah 1, Diakarta, Indonesia.

ABSTRACTS OF RADIOLOGICAL LITERATURE

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

HEAD

- DEE, P. M.: Benign hypertrophy of the masseters: a report of the radiological features in two cases. 258
- RITCHIE, G. MACL.: Hypophosphatasia: a metabolic disease with important dental manifestations. 258
- RICE-SIMONS, R. A. *et al.*: Extracranial occlusive vascular disease obscuring intracranial vascular lesions. 258
- HOHL, M., and BAKER, H. R.: The atlanto-axial joint: roentgenographic and anatomical study of normal and abnormal motion. . . . 258

NECK AND CHEST

- DI GUGLIELMO, L., and BERETTA, L.: The significance of laryngography with contrast medium in the study of chronic, nonspecific inflammation of the larynx. 259
- FERRARI, G.: Foreign bodies in the hypopharynx and esophagus (study of the radiologic morphology in 40 cases). 259
- SIMON, G.: Radiology and emphysema. 259
- REID, L., and MILLARD, F. J. C.: Correlation between radiological diagnosis and structural lung changes in emphysema. 260
- LANSING, A. M.: Radiological changes in pulmonary atelectasis. 261
- BOUCOT, K. R. *et al.*: Appearance of first roentgenographic abnormalities due to lung cancer. 261
- BRIGDEN, W., and ROBINSON, J.: Alcoholic heart disease. 261
- RAZZAK, M. A., HIGAZI, A.-R. M., and GUIRGIS, B.: Radiocardiography in rheumatic heart disease and its value in selection of cases for mitral commissurotomy. 262
- LYNCH, H. T. *et al.*: Complete transposition of great vessels: manifestation in two siblings confirmed at autopsy. 262
- HECKMANN, K.: Lateral tilt (frontodiagonal) roentgenography for showing superior mediastinum, trachea and aortic arch. 263

ABDOMEN

- CUMMACK, D. H.: The diagnosis of massive hematemesis: the role of radiology. 263
- PIRK, F., and VULTERINOVA, M.: The x-ray picture of the small intestine and impaired absorption. 263
- BOND, M. R., and ROBERTS, J. B. M.: Intussusception in the adult. 263
- BUCHAN, D. J., and HOUSTON, C. S.: Small bowel ulceration associated with enteric-

- coated potassium chloride and hydrochlorothiazide. 264
- MC ELWAIN, J. W., ALEXANDER, R. M., and MACLEAN, M. D.: Toxic dilatation of the colon in acute ulcerative colitis. 264
- HUNTER, G. A.: A case of annular pancreas presenting as a cause of acute intestinal obstruction in the neonate. 264
- ESSER, G.: Technique of percutaneous hilus-directed puncture of the spleen in splenoportography. 265

GYNECOLOGY AND OBSTETRICS

- MORGAN, J., and CADGER, D.: Arteriovenous fistula of the internal iliac artery and vein, related to trauma at childbirth. 265
- BARRON, L. R., HILL, R. O., and LINKLETTER, A. M.: X-ray pelvimetry. 266

GENITOURINARY SYSTEM

- ROSS, G., JR. *et al.*: High dosage excretory urography. 266
- ABDULHAYOGLU, S., and MARBLE, A.: Necrotizing renal papillitis (papillary necrosis) in diabetes mellitus. 266
- WILLIAMS, J. L., and FISHER, M.: The radioactive isotope renogram and urinary obstruction. 267
- LOKEN, M. K. *et al.*: Radioisotope renogram in kidney transplants. 267
- DE BERNARDI, E., and SPAZIANTE, G.: Recovery of the renal function after surgical intervention. 267
- PALUBINSKAS, A. J., PERLOFF, D., and WYLIE, E. J.: Curable hypertension due to renal artery lesions. 268
- KAISER, T. F., and FARROW, F. C.: Injury of the bladder and prostatic membranous urethra associated with fracture of the bony pelvis. 268

BLOOD AND LYMPH SYSTEM

- CHOI, J. K., and WIEDEMER, H. S.: Chyluria: lymphangiographic study and review of literature. 269
- KISHIMOTO, T. *et al.*: Lymphography in a patient with unilateral chyluria. 269

SKELETAL SYSTEM

- EECKELS, R., VINCENT, J., and SEYNHAEVE, V.: Bone lesions due to smallpox. 270
- GORTVAI, P., and ROPER, A.: Pott's disease with congenital anomalies of the spine. . . . 270
- DOWNS, W. J.: Multiple pseudocystic tuberculosis of bone. 270

ROENTGEN DIAGNOSIS

HEAD

DEE, P. M. Benign hypertrophy of the masseters: a report of the radiological features in two cases. *Clin. Radiol.*, Oct., 1964, 15, 347-349. (From: Department of Radiology, The Royal Victoria Infirmary, Newcastle upon Tyne, England.)

Since the first description by Legg in 1880 only approximately 40 cases of benign masseter hypertrophy have been reported in the literature. Two additional cases are described by the author, one of which was diagnosed roentgenologically.

The roentgenologic features are distinctive and consist of: (1) normal filling of the parotid gland; (2) lateral displacement of the parotid duct and the accessory part of the parotid gland in the presence of a roentgenologically normal parotid gland; and (3) lateral flaring of the angles of the mandible, thought to be due to the action of the hypertrophied muscles.—*Samuel G. Henderson, M.D.*

ITCHIE, G. MacL. Hypophosphatasia: a metabolic disease with important dental manifestations. *Arch. Dis. Childhood*, Dec., 1964, 39, 584-590. (From: Department of Dental Surgery, University College Hospital Dental School, London, England.)

The author has studied the teeth of 10 children suffering from hypophosphatasia. Particularly when the disease is severe and occurs during the time when dental tissues are being formed, there is hypoplastic deformity and crowding and early loss of teeth in both dentitions. Many deciduous teeth are lost prematurely with very little resorption of their roots. There is no compensatory early eruption of the permanent teeth. The permanent incisors, especially the uppers, become loose early and suffer periods of looseness and relative firmness. The jaws of these patients tend to be small, so that a high tooth/bone ratio results.

Treatment consists of good oral hygiene, good dietary habits and the use of fluoride compounds. Dentures should be provided to prevent overloading the teeth and to support those that are loose.—*Arthur E. Childe, M.D.*

RICE-SIMONS, RUTH A., VIAMONTE, MANUEL, JR., GARGANO, FREDIE P., and SPEAR, HAROLD C. Extracranial occlusive vascular disease obscuring intracranial vascular lesions. *South. M. J.*, Dec., 1964, 57, 1391-1395. (From: Departments of Neurology, Radiology, and Surgery, University of Miami School of Medicine, and Jackson Memorial Hospital, Miami, Fla.)

A patient is reported with a bleeding aneurysm of the basilar artery in whom the symptoms were erroneously attributed to occlusion of the innominate artery and the subclavian steal syndrome.

The patient had had severe right sided head and facial pain for 3 years. Approximately 1 year before referral to Jackson Memorial Hospital, he had a transient episode of left hemiplegia and right facial paralysis. Subsequently, neck pain and rigidity were present for several weeks.

Physical examination revealed no carotid or brachial pulses on the right. A transfemoral retrograde aortogram was obtained and revealed the characteristic findings of a subclavian steal syndrome secondary to occlusion of the innominate artery. The intracranial circulation was not studied.

He was referred to Jackson Memorial Hospital for correction of the occlusion of the innominate artery. Repeat studies of the vascular system included both the great vessels and the intracranial circulation. The occlusion of the innominate artery was confirmed as well as the subclavian steal syndrome. However, an aneurysm at the origin of the basilar trunk was also demonstrated. Because of the location of the lesion and the patient's asymptomatic status, repair was not considered indicated at that time.

The authors postulate that the patient's symptoms were secondary to bleeding of the intracranial aneurysm and that the subclavian steal syndrome was asymptomatic. They also stress that the status of the intracranial circulation should be evaluated before correction of extracranial occlusive vascular disease is undertaken.—*Barry Gerald, M.D.*

HOHL, MASON, and BAKER, H. R. The atlantoaxial joint: roentgenographic and anatomical study of normal and abnormal motion. *J. Bone & Joint Surg.*, Dec., 1964, 46-A, 1739-1752. (Address: Dr. Hohl, 435 North Roxbury Drive, Beverly Hills, Calif.)

The anatomy of the atlas and axis is reviewed. The fact that the articular surfaces of the lateral joints of the atlas and axis are convex and nonreciprocal is important, since it explains the roentgen findings on rotation.

Studies were made of the atlantoaxial motions using clinical material, a fresh anatomic specimen and cineroentgenography. The position of the odontoid in relationship to the lateral masses of the atlas and the relative positions of the lateral masses of the atlas and axis in normal cases are important as regards the significance of findings noted after injury.

The authors made the following observations:

1. Lateral bending alone without rotation at the atlantoaxial joint produced no lateral shift of the atlas on the axis.
2. Lateral bending to 15 degrees without rotation of the head produced atlantoaxial rotation in all but

1 patient. The spinous process of the axis rotated to the side opposite that of the lateral bending. There was articular facet offset and asymmetry in the paraodontoid spaces.

3. Lateral bending to physiologic limits showed an offset of the lateral masses of the atlas and axis ranging from 0-5 mm. on one side and a total right to left shift of 0-8 mm. (Only one patient had no shift.)

4. Rotary motion in proportion to the amount of rotation caused superimposition of the lateral joint surfaces on the side towards which the spine of the axis was directed, and the other lateral joint appeared narrowed.

5. Rotary motion alternately increased and decreased the apparent vertical height of the atlas and axis, substantiating vertical approximation as a normal motion.

6. Roentgenograms in flexion and extension showed an average of 1-2 mm. movement between the anterior arch of the atlas and the odontoid process.

The conclusion is reached that small degrees of asymmetry are common and there may appear to be lateral subluxation of the atlantoaxial joints due only to positioning or spasm. Lateral shift is a normal physiologic movement. The sliding motion is normal and not indicative of subluxation.—*Martha Mottram, M.D.*

NECK AND CHEST

DI GUGLIELMO, L., and BERETTA, L. Il significato della laringografia con mezzo di contrasto nello studio delle flogosi croniche aspecifiche della laringe. (The significance of laryngography with contrast medium in the study of chronic, nonspecific inflammation of the larynx.) *Radiol. med.*, Oct., 1964, 50, 946-959. (Address: Dott. Prof. Lucio Di Guglielmo, Policlinico, Pavia, Italy.)

In the present study, a systematic analysis of the laryngographic findings in chronic laryngeal inflammations is conducted.

Criteria have been established for precise diagnostic evaluation. The authors have observed that in diffuse chronic inflammatory processes, although there may be a marked thickening of the true and false vocal cords, laryngography in deep inspiration always demonstrates complete abduction of the cords and flattening of the glottis. On the contrary, in the presence of neoplastic infiltration, abduction of the cords is absent on the affected side.

Characteristic laryngographic findings have been demonstrated in 20 patients, including cases of diffuse hypertrophic states, ventricular eversion, polyps and verrucae.—*Anthony A. Blasi, M.D.*

FERRARI, G. Corpi estranei nell'ipofaringe ed esofago (studio di morfologia radiologica su

40 casi). (Foreign bodies in the hypopharynx and esophagus [study of the radiologic morphology in 40 cases].) *Ann. radiol. diag.*, 1964, 37, 151-176. (From: Reparto di Radiologia e Terapia Fisica, Ospedale Civile di Imola, Bologna, Italy.)

Roentgenography is the simplest, most innocuous and surest method of diagnosis and localization of foreign bodies in the hypopharynx and esophagus. The determination of the point of arrest and of the form and dimension of the foreign body is of invaluable assistance to the endoscopist.

The author is in accord with many other investigators as to the roentgenologic method to be used. After a preliminary "flat" roentgenogram is obtained, additional roentgenograms are taken following the administration of a semifluid opaque bolus and the ingestion of a few mouthfuls of water.

The author describes different patterns of roentgenographic images which are produced at the level of the foreign body following a barium swallow: (1) "Pincer" blockage; (2) "drop" blockage; (3) "circumscribed blockage" in which there is a widening of the barium column with central or marginal defect or defects; and (4) "target" blockage.

The importance of the administration of water following the barium swallow is stressed. A thin layer of barium adheres to the foreign body, coats it and brings it into relief.—*Anthony A. Blasi, M.D.*

SIMON, GEORGE. Radiology and emphysema. *Clin. Radiol.*, Oct., 1964, 15, 293-306. (From: St. Bartholomew's and Brompton Hospitals, London, England.)

In the Ciba Symposium of 1959 emphysema was defined as abnormally large air spaces beyond the terminal bronchiole. A classification of Lynne Reid in 1964 of the various types of emphysema lists under two main headings: (a) emphysema without airways obstruction, and (b) emphysema with airways obstruction. The latter is further classified as (1) reversible airways obstruction, and (2) irreversible airways obstruction. The author, in this article, discusses gross widespread emphysema with air trapping (airways obstruction), either idiopathic in origin or on the basis of chronic bronchitis.

The criteria for the roentgen diagnosis of gross widespread emphysema chosen by Simon and Galbraith in 1954 were as follows: (1) an excess of air in the lungs with air trapping demonstrated by a low, flat and poorly moving diaphragm and a large retrosternal area; (2) certain changes in the appearance of the cardiac contour and lung vessels, particularly a narrow vertical heart, a large pulmonary trunk, and a combination of large hilar vessels with small lung vessels; and (3) the presence of bullae or bullous areas demonstrated by localized avascular relatively translucent areas.

The diaphragm can be considered low if the central portion of the right dome of the diaphragm on inspiration lies at or below the level of the 7th rib anteriorly (below the level of the 6th rib anteriorly in 1 patient of hypersthenic habitus). The range of movement between full inspiration and full expiration is diminished—usually less than 3 cm. The depth of the translucent zone behind the sternum often measures 3 to 5 cm., compared with the normal of 2 to 3 cm. This radiolucent zone tends to extend lower in emphysema than in a normal person. The phenomenon of the narrow vertical heart is probably in part the result of the low position of the diaphragm, allowing the heart to become more dependent and therefore narrower. One often sees in emphysema a slight or moderate prominence of the left heart border below the aortic knuckle (aortic knob). The combination of relatively large hilar vessels with relatively small mid-lung vessels is a very important finding in general emphysema. In a normal person large hilar vessels are associated with relatively large lung vessels, and, conversely, small hilar vessels with small lung vessels. In emphysema large hilar vessels lead almost at once to apparent relatively small lung vessels. A third criterion for the diagnosis of emphysema is based on a combination of the first two, consisting of an excess of air giving a localized relatively transradiant area, together with few or no vessel shadows within it, indicating a bulla or bullous area. A bulla may be demonstrated by a hair-line shadow.

Whenever widespread emphysema has been diagnosed on the basis of the foregoing criteria and morbid anatomic studies have been possible, gross widespread emphysema with air obstruction has invariably been found. When the emphysema is due to chronic bronchitis, there will also be cough and sputum. Respiratory function studies have invariably revealed severe airways obstruction, more or less irreversible, in those patients with the roentgenologic emphysema pattern. Once an obvious emphysema pattern can be seen on the roentgenogram, the prognosis is rather poor.

A normal roentgenogram almost definitely excludes gross widespread emphysema. The milder grades of emphysema, even when they are uniform and widespread throughout the lungs, do not cause abnormalities on the chest roentgenogram, but severe grades if widespread invariably give roentgenologic changes. The roentgenographic evidence may be misleading unless carefully correlated with the other findings, as when gross widespread emphysema is associated with cor pulmonale, either in clinical failure or on the verge of this. In this case one may find a large heart with relatively large lung vessels and a diaphragm of almost normal shape.

There is doubt as to the development of emphysema, whether it arises as a complication of chronic bronchitis or is idiopathic. Only about one-third of patients with chronic bronchitis eventually develop

gross emphysema. The emphysema pattern is commonly first seen between the ages of 45 to 60 years. The usual course of events after the emphysema pattern has been seen is that further roentgenograms over the years are unchanged, but the dyspnea gets worse. Many patients with severe dyspnea and evidence of irreversible air-ways obstruction have a normal chest roentgenogram. The cause of the symptoms may be bronchospasm, bronchial mucosal edema or airway obstruction from an excess of mucus in the more peripheral airways.

The author concludes that the roentgenologic emphysema pattern appears to be a reliable indication of severe widespread emphysema and carries a poor prognosis. Clinically, a patient with cough, sputum and dyspnea from chronic bronchitis may be indistinguishable from a similar patient with complicating emphysema.—*Samuel G. Henderson, M.D.*

REID, LYNNE, and MILLARD, F. J. C. Correlation between radiological diagnosis and structural lung changes in emphysema. *Clin. Radiol.*, Oct., 1964, 15, 307-311. (From: Institute of Diseases of the Chest and The Brompton Hospital, Fulham Road, Brompton, London, S.W.3, England.)

The authors give a detailed correlation of the pathologic severity of the emphysema and the roentgenologic findings. The roentgenographic appearances were compared with the autopsy findings in 40 cases, of whom 37 had chronic bronchitis, diagnosed on the basis of cough and sputum for more than 3 years. Two of the 40 cases had idiopathic emphysema, and 1 had primary pulmonary hypertension.

The essential feature of the emphysematous lungs was a loss of substance, manifested first by an increase in the size of the air spaces, and secondly by retraction of the alveoli below the airways and vessels on the cut surface. The pathologic grading was made on the basis of the size of the air spaces and on the degree of retraction of the alveoli. In Grade I the air spaces are abnormally large, up to 1 mm. in diameter, but retraction is slight. In Grade II retraction is striking, and the air spaces are larger than in Grade I. In Grade III the air spaces may be up to 5 mm. in diameter, and in Grade IV there are "holes" larger than 5 mm. in diameter.

Grade III is the minimum severity of emphysema producing roentgenographic changes. When half to two thirds of the largest lung slice shows Grade III changes, the roentgenographic appearance will be that of widespread emphysema.

The following roentgenographic diagnoses of the 40 subjects were made using Simon's criteria: no emphysema, 19; widespread emphysema, 10; localized emphysema, 9; and "possible slight" emphysema, 2.—*Samuel G. Henderson, M.D.*

LANSING, ALLAN M. Radiological changes in pulmonary atelectasis. *A.M.A. Arch. Surg.*, Jan., 1965, 90, 52-56. (Address: 511 S. Floyd Street, Louisville, Ky. 40202.)

The clinical picture of pulmonary atelectasis with fever, tachycardia, rapid respiratory rate and other features is often seen with negative roentgenologic findings. This stimulated the experimental investigation of the time lapse factor between the actual obstruction of a bronchus and the roentgenologic appearance of atelectasis. The effect of changing the gas in the alveoli behind the obstruction from air to 100 per cent oxygen was also noted.

Dogs were used and in one group obstruction of one of the main stem bronchi was produced by inserting a cotton plug while in a second group obstruction was produced by actually tying off either the right middle lobe bronchus or both the right middle and lower lobe bronchi. Chest roentgenograms then were taken at periodic intervals up to and sometimes including a 96 hour period. A portion of each group of bronchial obstructions was hyperventilated with room air before the obstruction was placed and the remainder of the group was ventilated with 100 per cent oxygen.

Those animals with room air in the distal bronchial tree behind the cotton plug began to show roentgenologic evidence of atelectasis in 5 to 48 hours with a median time of 12 hours. Those with oxygen present began to show atelectasis in from 1 to 12 hours with a more complete collapse of the lung being noted. Removal of the cotton plugs resulted in reexpansion of the lung in 3 to 5 hours with complete recovery in all cases.

Ligation of the bronchial tree produced similar results. The clinical appearance of atelectasis with increased temperature, heart rate, and respiratory rate was observed with roentgenologic changes developing in from 1 to 5 hours in the oxygenated cases and in 12 to 48 hours in those with only room air present. This would indicate that postoperative inflation of the lungs should be carried out with room air rather than 100 per cent oxygen.

In conclusion, it is noted that roentgenologic evidence of pulmonary atelectasis distal to a bronchial obstruction is not seen for at least 5 hours or more. If 100 per cent oxygen is trapped in the alveoli distal to the obstruction, massive pulmonary collapse develops more rapidly. The clinical picture of pulmonary atelectasis deserves treatment despite negative roentgenologic findings.—Richard R. Kinzer, M.D.

BOUCOT, KATHARINE R., COOPER, DAVID A., WEISS, WILLIAM, and CARNAHAN, WILLIAM J. Appearance of first roentgenographic abnormalities due to lung cancer. *J.A.M.A.*, Dec. 28, 1964, 190, 1103-1106. (Address: Dr. Boucot, 311 Juniper Street, Philadelphia, Pa. 19107.)

A total of 6,137 men aged 45 years or more was followed by semiannual photofluorograms and symptom questionnaires for 8 to 10 years. Seventy-six histologically confirmed carcinomas developed during this period and in 45 cases the lung cancer appeared within 9 months of a normal photofluorogram. A special study of this latter group showed that about two thirds of the cancers arose peripherally and that the first abnormalities noted were extremely variable. Nine were solitary nodules, 5 were frank masses, 1 was a homogeneous irregular density and 12 were small irregular infiltrates. The first roentgenologic findings in the cancers of central origin varied from minor changes in the hilar area, often difficult to detect, to obvious atelectasis.

Throughout most of the period dual reading was carried out and a third reader read those photofluorograms on which the first two readers disagreed.

The authors stress the importance of over-reading chest roentgenograms for cancer as the first abnormalities are highly variable and often unimpressive. Even expert readers in this series failed to agree upon the significance of the first abnormality in almost half of the "new" cancer cases.—Arthur E. Childe, M.D.

BRIGDEN, WALLACE, and ROBINSON, JOHN. Alcoholic heart disease. *Brit. M. J.*, Nov. 21, 1964, 2, 1283-1298. (Address: Dr. Brigden, The London Hospital, London, England.)

This report is based on 50 patients seen at the London Hospital and the National Heart Hospital between 1952 and 1963. The criteria for their inclusion in this series were a long history of a very high consumption of alcohol and evidence of heart disease which was not due to hypertension, coronary artery disease, valvular disease, or any other disorder known to cause heart disease.

With one exception all were men; their ages ranged from 33 to 72 years and most were between 45 and 65 years. All were heavy drinkers continuously for more than 10 years. One-third drank beer only, an average consumption being 15 pints a day; one-quarter consumed only spirits at a rate of one bottle or more a day; and the remainder drank beer and spirits in large quantities. Their occupations were of interest; 17 worked in the liquor trade or in close association with it, such as barmen, waiters, etc. All had been examined on several occasions and had had roentgenologic examination of the chest, electrocardiograms, urinalyses, hemoglobin estimations, leukocyte counts, and many other investigations, including liver function test and serum protein analyses. Twenty-five of these 50 patients have died. Nine were examined at necropsy and none showed significant coronary disease. The majority (19) died of heart failure, 2 died of pulmonary embolism, and 1 of cancer; the cause of death was uncertain in 3. Thirteen of the series were included in a previous study on non-coronary myocardial disease (Brigden, 1957).

The clinical features were evidence of cardiovascular symptoms appearing slowly over 1 to 2 years with gradual development of heart failure in the majority of patients; however, in some the onset was abrupt because of the development of irregular rhythm or an attack of paroxysmal nocturnal dyspnea. All had dyspnea on effort, which was thus the commonest symptom. In some patients this had been erroneously attributed to associated features such as obesity or bronchitis. Nearly half had had one or more attacks of paroxysmal dyspnea.

Tachycardia was present whether there was sinus rhythm or not. Ectopic beats were common and were often multifocal in origin. Twenty-five patients had atrial fibrillation at some time. The atrial fibrillation was more common in the older patients in the series. Chronic cough was common.

Cardiac enlargement was seen on roentgenologic examination in every patient. The left heart was primarily affected. The roentgenologic signs depended on the degree of myocardial damage and its resulting hemodynamic disturbance rather than the etiology of the process.

Some degree of pulmonary venous congestion was common but pulmonary edema was not seen. Small pleural effusions were common as in other forms of congestive cardiac failure. Only 2 had recognized episodes of pulmonary infarction with associated roentgenologic features.—*Peter C. Truog, M.D.*

RAZZAK, MUHAMMAD ABDEL, HIGAZI, ABDEL-RAHMAN M., and GUIRGIS, BADIE. Radiocardiography in rheumatic heart disease and its value in selection of cases for mitral commissurotomy. *J. Nuclear Med.*, Nov., 1964, 5, 851-863. (From: University of Cairo, Faculty of Medicine, Division of Nuclear Medicine, Cairo, Egypt.)

The use of surgery in rheumatic valvular heart disease has made the accurate diagnosis of the site, type and grade of the cardiac lesion of great importance.

In this report of 76 patients, 64 had evidence of a valvular lesion and the remaining 12 suffered from rheumatic fever with or without active carditis. All had clinical, roentgenologic, radiocardiographic and electrocardiographic examinations. In some cases of mitral stenosis the results of radiocardiography were correlated with operative commissurotomy findings.

A dose of 12 μ c of I^{131} serum albumin was rapidly injected intravenously with the patient in the supine position. The radioactive bolus traveling through the cardiac chambers was detected by scintillation counters, one scintillator pointing towards the center of the roentgenographic heart image and the other being centered just medial to the apex beat. The information was measured by 2 synchronized count rate meters and was recorded on a dual strip chart. The first detector registered the common radio-

cardiographic tracing, whereas the second helped define the peak of left heart radioactivity.

The normal radiocardiogram is made of two waves, R and L, representing the right and left sides of the heart, respectively. R is higher in amplitude than L, which is longer in duration, since radioactivity is diluted into the pulmonary bed before reaching the left side of the heart. The ascending limb of each wave stands for filling, whereas the descending limb represents emptying of the corresponding cardiac chambers. A dip between the two curves is caused by passage of radioactive material from beneath the two detectors into the lesser circulation. Thus the time interval between the peaks of R and L waves represents roughly the pulmonary circulation time. The mean value which approximates closely the real mean pulmonary circulation time can be obtained by means of a formula, in the interval between the minimum and maximum times of a common radiocardiographic curve.

The different valvular lesions of rheumatic valvular heart disease were found to have characteristic radiocardiographic patterns, depending on the site and degree of the lesion and the presence of an enlargement of the various cardiac chambers, together with the degree of pulmonary hypertension.

The importance of radiocardiography in selection of cases for mitral commissurotomy is stressed, since this technique is easy to perform and readily gives an idea of the width of the mitral orifice as well as of the degree of pulmonary artery pressure.—*Richard Pfister, M.D.*

LYNCH, H. T., GRISSOM, R. L., MOORING, P., and KRUSH, A. Complete transposition of great vessels: manifestation in two siblings confirmed at autopsy. *J.A.M.A.*, Dec. 14, 1964, 190, 969-972. (Address: Dr. Lynch, 44th and Dewey Avenue, Omaha, Neb. 68105.)

The authors describe 2 siblings with complete transposition of the great vessels confirmed at autopsy, the second reported occurrence of the anomaly in siblings.

Other families have been described in which complete transposition of the great vessels occurred in one member and other congenital heart defects in another member of the family. Complete transposition of the great vessels has also been shown to occur more frequently in males. The incidence of any congenital heart disease among siblings of an affected propositus has been stated to increase by 5 to 7 times above the expected incidence in the general population; however, this may be related partially to selection.

Because of these factors, the authors postulate a recessive mode of genetic transmission in the 2 patients described. The question is raised whether the same allele responsible for the tetralogy of Fallot and

other truncoconal malformations could cause complete transposition of the great vessels in a different environment.—Barry Gerald, M.D.

HECKMANN, K. Die frontale Diagonalaufnahme. (Lateral tilt [frontodiagonal] roentgenography for showing superior mediastinum, trachea and aortic arch.) *Röntgen-Blätter*, Dec., 1964, 17, 551-558. (Address: Hapsburgerplatz 1, 8 München 13, Germany.)

Conventional roentgenographic methods are inadequate for showing the trachea, superior mediastinum and aortic arch. In the sagittal projection, the trachea is superimposed on the thoracic spine; in the lateral projection, it is obscured by the overlapping shoulders. Oblique projections are more helpful, although the shoulders cannot always be entirely eliminated. Even then, the overlapping pulmonary structures fail to provide a sufficient contrast density. Laminagrams cannot replace the conventional summation technique because the trachea is not parallel to the table top in either the sagittal or the lateral projection. Lateral tilt (frontodiagonal) roentgenograms, on the other hand, have proved adequate to show the trachea in two directions at right angle to each other.

The technical details are as follows: In the erect posture, the patient tilts his body to the right as far as possible, so that the right shoulder leans against the lower border of an erect grid cassette. The left arm is elevated with the hand resting against the occiput. The central beam is directed toward the left midaxillary line at the level of the nipple at such an angle that the exit beam is in the right supraclavicular fossa. When the patient is flexible, the central beam usually is horizontal; but when he is rigid, an upward tilt of the roentgen tube becomes necessary. Because of the thickness of the parts, the author uses a kilovoltage of 125 kvp. Transmitted aortic pulsation of the lower trachea is negligible so that flash exposures are not necessary.

With this technique, the trachea in its entire length can be visualized. In the right lateral view, the left stem bronchus presents an orthograde projection. Barium coating of the adjacent esophagus is recommended to enhance the contrast of the posterior tracheal wall.—Ernest Kraft, M.D.

ABDOMEN

CUMMACK, D. H. The diagnosis of massive hematemesis: the role of radiology. *Brit. J. Surg.*, Oct., 1964, 51, 744-746. (From: Western General Hospital, Edinburgh, Scotland.)

Upper gastrointestinal study in patients with massive hematemesis will define the site of bleeding in approximately 60 per cent of cases. Barium is found to be far superior to gastrograffin, and no operative

or postoperative complications ensued due to barium. Examination with the mobile unit on the ward is not adequate, and no additional hazard to the patient follows examination in the X-Ray Department provided thoughtful care is taken.

Twenty of 23 patients with massive hematemesis studied over a 6 month period had correct roentgenologic diagnoses, proven at surgery or postmortem examination. The position of blood clot frequently gave an indication of the site of the lesion. Where the stomach was involved, considerable defect was seen in the stomach and little in the duodenum, and in duodenal lesions the reverse was the case. Meticulous examinations occasionally revealed an artery in the base of the demonstrable ulcer.—Mark D. Reiss, M.D.

PIRK, F., and VULTERINOVA, M. The x-ray picture of the small intestine and impaired absorption. *Radiol. clin.*, 1964, 33, 249-267. (From: Institute of Human Nutrition, Prague, Czechoslovakia.)

The authors compared the roentgen findings of small bowel examinations with their clinical and biochemical studies to see if reliable roentgen evidence of impaired absorption could be found.

The patients were evaluated clinically and using laboratory methods to appraise the absorption capacity of the intestine. The roentgen evaluation was based upon a combination of findings, including the width of the small bowel loop as a measure of tonicity (the bowel gas pattern); the appearance of the mucosal pattern; the presence of hypersecretion; and segmentation and flocculation. The presence or absence of these findings and their extent enabled the authors to place each case in one of 4 categories of normal and increasing severity.

The patients examined formed 4 groups: normal volunteers; routine follow-up gastrectomy patients; gastrectomy patients who were having some complaints and were thus seen in the department as being symptomatic; and a small group of malabsorption syndrome patients.

The results of the study showed that all of the "normal" volunteers had normal roentgen findings. Also, the gastrectomy patients with abnormal roentgen studies had a lower than average body weight and more frequent clinical signs of hypovitaminosis and anemia. Segmentation and flocculation were not found to be helpful signs, due to the use of non-flocculating barium.

Comparison of the fat and nitrogen balance studies and the absorption tests with the roentgen findings demonstrated a close correlation between pathologic roentgen appearance and the malabsorptive state.—Lawrence U. Cookson, M.D.

BOND, M. R., and ROBERTS, J. B. M. Intussusception in the adult. *Brit. J. Surg.*, Nov.,

1964, 51, 818-825. (From: Royal Infirmary, Sheffield, England.)

Various authors list adult cases of intussusception as occurring in only approximately 5 per cent of all cases. The form seen in the adult is almost exclusively secondary to a bowel lesion, in contrast to the more frequent infant intussusception which is "primary," although much speculation has been given to the role of lymphoid hyperplasia and ileocecal lymphadenopathy in the etiology of intussusception in the infant and child.

Intussuscepting lesions in the adult include: small intestine—benign neoplasm, malignant neoplasm, both primary and metastatic, Meckel's diverticulum; appendix—rarely with a mucocoele; large intestine—benign and malignant neoplasms.

The universal complaint was colicky abdominal pain, commonly associated with rectal bleeding.

Abdominal survey roentgenogram usually shows evidence of intestinal obstruction, and commonly outlines the tumor as a soft tissue mass. Barium enema study is diagnostic and shows the typical changes of intussusception. Although the intussusception is at times completely reduced at the time of the barium enema examination, laparotomy for resection of the primary lesion is recommended.

Over a 7 year period the incidence and site of 16 cases of intussusception in the adult were compared with the incidence and distribution of small and large bowel tumors. Three of 27 small intestinal tumors presented as an intussusception. Although colon neoplasms were much more frequent, the transverse colon, which was the next most frequent source of intussusception relative to its share of tumors, had less than half the incidence of the small intestine. Adequate motility of the bowel is an obvious necessity for the development of an intussusception, and thus those reaches of the intestine which have a long mesentery, such as the small intestine, are far more frequent sites of intussusception than the areas, such as the descending colon, which have no mesentery.—*Mark D. Reiss, M.D.*

BUCHAN, DOUGLAS J., and HOUSTON, C. STUART. Small bowel ulceration associated with enteric-coated potassium chloride and hydrochlorothiazide. *Canad. M. A. J.*, Jan. 23, 1965, 92, 176-179. (From: Departments of Medicine and Radiology, University of Saskatchewan, and University Hospital, Saskatoon, Saskatchewan, Canada.)

During the past year 3 cases of small bowel obstruction due to localized ulceration, inflammation, and stenosis were seen at the University Hospital, Saskatoon, Saskatchewan. Considerable difficulty in reaching a diagnosis was observed although the patients did have signs of incomplete small bowel obstruction. At laparotomy, the lesions were found 5

inches above the ileocecal valve, 2 feet above the ileocecal valve, and about half way down the small bowel, respectively. Each of these 3 patients had taken a preparation of hydrochlorothiazide and potassium chloride with an enteric coating (Hydro-Diuril-Ka). The medication, given for mild hypertension, was taken for 26 days, 28 days, and 17 months, respectively.

Two other series of similar cases have been reported, but to date there have been no reports of ulceration with the use of non-enteric-coated preparations of hydrochlorothiazide and potassium chloride. The composition of the enteric coating itself is suspect, and from present information, the lesions are produced only when hydrochlorothiazide, potassium chloride, and an enteric coating are combined.—*Richard E. Kinzer, M.D.*

McELWAIN, JACK W., ALEXANDER, RICHARD M., and MACLEAN, M. DOUGLAS. Toxic dilatation of the colon in acute ulcerative colitis. *A.M.A. Arch. Surg.*, Jan., 1965, 90, 133-142. (Address: Dr. McElwain, 4277 Hemstead Turnpike, Bethpage, N. Y.)

Toxic dilatation of the colon, occurring in the initial phase of ulcerative colitis or as an acute exacerbation of the disease, is one of the most serious complications. Since the cause is not clear and the treatment is not well established, the authors have elected to report their experience in 9 such cases.

Four patients in this series had been given barium enemas within a 2 week period prior to the appearance of the toxic dilatation. Others have noted this—"more than casual"—relationship and it is suggested that barium enema examinations *should not* be performed in the acute phase of ulcerative colitis. Opiates and anticholinergics have been blamed for the production of dilatation and should probably be avoided also.

Toxic dilatation is an acute medical and surgical emergency and its presence may not always be obvious. Repeated flat roentgenograms of the abdomen at 12 to 24 hour intervals are recommended. Conservative treatment beyond 2 to 5 days is not advised unless the white blood cell count drops and the dilatation decreases. The operation of choice is the one stage ileocoloproctectomy with permanent ileostomy.

Summary of authors' cases includes symptoms, laboratory findings, and medical and surgical management.—*Richard E. Kinzer, M.D.*

HUNTER, G. A. A case of annular pancreas presenting as a cause of acute intestinal obstruction in the neonate. *Brit. J. Surg.*, Nov., 1964, 51, 875-876. (From: St. James's Hospital, Balham, England.)

Annular pancreas presenting as intestinal obstruction

tion in the neonatal period has rarely been reported in the English literature; therefore, a single typical case is reviewed.

Annular pancreas results because of failure of the anterior pancreatic bud to follow the rotation of the foregut and consequently a ring-like configuration of the fused ventral and dorsal pancreatic buds occurs. Such a pancreas, if symptomatic in the neonatal period, results in high intestinal obstruction. Bile stained emesis is common and jaundice may occur.

Roentgenologic examination of the upright abdomen reveals the "double bubble" appearance of the dilated stomach and proximal duodenum. Gas shadows are absent in the remainder of the intestine. Aspiration of the stomach contents via a nasogastric tube followed by the insufflation of 30 cc. of air into the stomach will aid in the roentgenologic demonstration of the high obstruction. Oral barium examination is contraindicated because of the possibility of aspiration. The value of barium enema examination is not discussed.

The differential diagnosis includes duodenal atresia, pyloric stenosis, duodenal volvulus, para-duodenal hernia and intestinal malrotation.

Seventy per cent of the reported cases cited had associated anomalies including duodenal atresia, tracheoesophageal fistula, Meckel's diverticulum, intestinal malrotation, imperforate anus, cleft palate, congenital heart disease, and mongolism. The mortality rate is reported as 45 per cent in these cases and the associated abnormalities were a major factor in this figure.

The danger of fistula or pseudocyst formation precludes division of the pancreatic ring. In addition, this procedure may not relieve the obstruction because of the possible intermingling of the duodenal musculature and pancreatic tissue. Duodenojejunostomy is the treatment of choice. If a sufficient length of duodenum cannot be mobilized, gastrojejunostomy is the alternative treatment; however, this does not always decompress the proximal duodenum and complications may occur in later life.—*John T. Underberg, M.D.*

ESSER, G. Zur Technik der perkutanen hilär gerichteten Milzpunktion bei der Splenoporthographie. (Technique of percutaneous hilus-directed puncture of the spleen in splenoporthography.) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Nov., 1964, 101, 495-500. (Address: Chir. Univ.-Klinik, 53 Bonn-Venusberg, Germany.)

Percutaneous splenoporthography can nowadays be done more accurately with the aid of image intensification and television monitoring. Thus the hazards of a "blind" procedure are eliminated. Anesthesia, however, with controlled respiration still remains a prerequisite. The indications for its use are portal hypertension and presence of gastroesophageal vari-

ces. Ascites, suggesting hepatic decompensation, represents a contraindication. Marked meteorism may also impair the results of the procedure.

An improved technique is described in detail.

The patient is placed on the radiographic table in supine position with the xyphoid process in the center. A preliminary roentgenogram is made to determine proper positioning, also the size and location of the spleen. Percussion is done to find the maximum dullness of the spleen. The puncture needle is introduced at the ninth or tenth intercostal space in midaxillary line. Its point is directed toward the hilus of the spleen under fluoroscopic control. The needle has to overcome the resistance of the splenic capsule and is guided a predetermined distance toward the splenic vein. This is followed by a preliminary test injection of 5 to 10 cc. of the contrast material.

One hundred and twenty cases were examined with this improved technique. In several cases, a subsequent laparotomy disclosed very little intraperitoneal hemorrhage with a maximum of 60 cc. in 1 case only.

The chief advantages of the improved technique are short anesthesia, more accurate pressure reading, and better filling of the portal and collateral vessels; also prevention of intrasplenic storage of contrast material and absence of complications. Perforation of a larger vessel, however, has to be guarded against. This can be avoided by the subtle sensation of elastic resistance offered by the vessel wall.—*Ernest Kraft, M.D.*

GYNECOLOGY AND OBSTETRICS

MORGAN, JOHN, and CADGER, DOUGLAS. Arteriovenous fistula of the internal iliac artery and vein, related to trauma at childbirth. *Canad. M. A. J.*, Oct., 1964, 91, 920-925. (Address: 62 Academy Medical Building, 1812 4th Street, Calgary, Alberta, Canada.)

The authors report 3 cases of arteriovenous fistula involving the internal iliac artery and vein and describe the clinical syndrome associated with this abnormality. All 3 cases were women who had pregnancy associated with delivery of large babies or long and difficult labor.

Each patient had shortness of breath and/or fatigued easily on exertion; 2 of the 3 had large hearts. One had complained that the leg on the side later to develop the fistula had "let her down" during the last trimester of her pregnancy. All 3 exhibited a loud bruit over the sacrum and 2 had bruit over the pubis as well.

The 3 cases were operated and correction of the fistulae resulted in relief of fatigue and shortness of breath. Due to the great number of contributing vessels, one fistula had to be corrected by three approaches.

Based on experimental investigation, the authors believe that a fistula must be over $\frac{1}{2}$ inch in diameter before clinical symptoms become manifest.

All fistulae were demonstrated by aortography. The ages of the patients were from 34 to 41 years.

The trauma associated with childbirth experienced by all 3 patients was assumed to be the chief contributing factor to the formation of the arteriovenous fistulae between the internal iliac artery and vein.—*Sam W. Campbell, M.D.*

BARRON, L. R., HILL, R. O., and LINKLETTER, A. M. X-ray pelvimetry. *Canad. M.A.J.*, Dec. 5, 1964, 91, 1209-1212. (From: Lachine General Hospital, Montreal, Quebec, Canada.)

The authors conclude that pelvimetry is of real value to the obstetrician in cases of definite bone disproportion when combined with the clinical appraisal of the patient, but in the borderline case radiology is not of much help, and the proper management in such cases is a good trial of labor.

The results of roentgenographic pelvimetry performed on 66 pregnant women, with a view to predicting the outcome of pregnancy, were reviewed.

The authors believe that pelvimetry in the later stages of pregnancy has no ill effects on the unborn baby or the mother. It is emphasized that the pelvic assessment should be made jointly by the radiologist and obstetrician.—*Eugene J. McDonald, M.D.*

GENITOURINARY SYSTEM

ROSS, GILBERT, JR., WILSON, WILLIAM J., ROARDS, VICTOR L., JR., and THOMPSON, IAN M. High dosage excretory urography. *J. Urol.*, Dec., 1964, 92, 728-731. (From: Section of Urology and Department of Radiology, University of Missouri School of Medicine, Columbia, Mo.)

A study of 67 urograms of 50 patients who had been given 30 cc. of 50 per cent hypaque showed that no patient had a truly satisfactory excretory urogram if the serum creatinine value exceeded 3.5 mg. per cent. Above 3 mg. per cent, patients with active obstruction often had good visualization while patients without obstruction frequently did not. The blood urea nitrogen in patients with satisfactory urograms was significantly higher in relation to the serum creatinine in the presence of obstruction than when renal function was stable. For this reason, the serum creatinine seems to have some slight advantage in predicting the outcome of excretory urography in the patient with obstruction.

A review of medical records and roentgenograms of adult patients with known reduction in renal function indicates that administration of doses of 50-60 cc. of radiopaque urographic material considerably improves visualization of the intravenous

pyelograms if there is evidence of depletion of renal function as indicated by a creatinine level consistently higher than 1.5 mg. per cent or a blood urea nitrogen higher than 25 mg. per cent. In 37 patients with a creatinine level of less than 2.5 mg. per cent, 30 cc. of radiopaque material produced 84 per cent successful intravenous pyelograms and in 13 patients with similar creatinine levels, 50-60 cc. of radiopaque material produced 85 per cent successful pyelograms. At blood creatinine levels of 2.5-3.5 mg. per cent, the intravenous pyelograms showed 80 per cent success with 60 cc. of radiopaque material compared to 60 per cent with 30 cc. At blood creatinine levels of 3.5-4.5 mg. per cent, there was an even greater spread. Patients injected with 60 cc. of radiopaque material had 40 per cent successful pyelograms, whereas with 30 cc. none showed adequate visualization of the excretory passages in the urinary tract.

Because of these facts and because there seemed to be very little increase in the hazard of using higher doses of intravenous radiopaque material, the authors now use 50-60 cc. of intravenous hypaque or conray routinely in adult patients. Laminagraphy is occasionally valuable if the creatinine levels are above 4 mg. per cent.

The intravenous radiopaque material is injected slowly in the usual manner prior to the film examination. More than 1,500 high dosage excretory urograms have been performed without serious reaction and no apparent increase in the number of minor reactions has occurred.

The authors include 3 tables and 1 composite illustration of intravenous urograms.—*George W. Chamberlin, M.D.*

ABDULHAYOGLU, SEFIK, and MARBLE, ALEXANDER. Necrotizing renal papillitis (papillary necrosis) in diabetes mellitus. *Am. J. M. Sc.*, Dec., 1964, 248, 623-632. (From: New England Deaconess Hospital [Joslin Clinic Service], Boston, Mass.)

The authors report 14 cases of necrotizing renal papillitis (papillary necrosis) in diabetic patients; 13 of these patients died. The surviving patient was found to have renal calculi in the same kidney 31 months after having had necrotizing renal papillitis.

The clinical course and the laboratory findings of the 13 patients revealed 3 different types: (a) an acute fulminating type; (b) a subacute type; and (c) a type in which the condition was an incidental finding at necropsy.

The authors believe that due to the frequent association of pyelonephritis and renal vascular disease with necrosis of the papillae in diabetic patients, these are the two major contributing factors in the development of the lesions.

Necrotizing renal papillitis is considered by many to be a form of pyelonephritis. It is generally accepted

that normally the papillae have less blood supply than the main portion of the medulla of the kidney. The widely held view is that ischemia of the papillae is the chief pathogenic factor.

In laboratory animals, the lesion has been produced by ligating the renal vein in rats, by ligating one ureter of rabbits followed by intravenous injection of a pathogenic organism and also by merely ligating the ureter of mongrel dogs.

The diagnosis of necrotizing renal papillitis has most often been made at postmortem examinations. The authors state that only 4 of their 13 fatal cases were diagnosed before death: 3 of these were of the acute fulminating type and the fourth of the subacute type. In their 13 cases, 2 had excretory urography; retrograde pyelography was done in 2. None of the authors' patients had obstructive uropathy, which in the past was accepted as one of the most important etiologic factors. The involvement can be unilateral or bilateral. Six of the patients showed the right kidney to concentrate and excrete dye better than the left kidney.—*Peter C. Truog, M.D.*

WILLIAMS, J. L., and FISHER, M. The radioactive isotope renogram and urinary obstruction. *Brit. J. Surg.*, Nov., 1964, 51, 825-827. (From: Department of Urology, Royal Hospital, Sheffield, and Regional Medical Physics Department, Sheffield, England.)

Thirty-eight patients, the majority having unilateral hydronephrosis, were studied by renography; 20 μ c of I^{131} hippuran was administered several hours after a blocking dose of Lugol's solution. The apparatus and method used were similar to those previously described. The rate of I^{131} hippuran excretion was substantially reduced only in bilateral hydronephrosis and consequently the radiation dose to the patient was generally estimated to be small.

The normal curve recorded following intravenous injection of hippuran I^{131} consists of a rapid rise to a maximum in 3-5 minutes and followed by a slower decline. The initial upward curve represents vascular flow and tubular secretion. The maximum height of the curve is lowered by diuresis and vasoconstriction and increased by vasodilatation, but the normal configuration is maintained.

In hydronephrosis the curve continues to rise above the normal peak due to an accumulation of hippuran and may not fall even after 1 hour. The degree of function of the kidney can be assessed by comparison of the normal and abnormal sides. In a normally functioning hydronephrotic kidney the initial rise is similar to the opposite side, whereas in a less well functioning kidney the curve rises more gradually. Even a minimal rise in the curve indicates renal function, as in a nonfunctioning kidney the curve falls due to the declining blood activity.

The time of fall in the curve cannot be correlated with renal function as several factors, including the

patient's degree of hydration, influence the fall-off. Also, the presence of a dilated renal pelvis, into which the hippuran is excreted and diluted by already present urine, masks the rate of urine flow even if it is normal, and so the decline in the curve is delayed.

This factor is also of importance in the postoperative evaluation of the kidney because, should the dilated pelvis persist after relief of the obstruction, the tracing will still show an "obstructive pattern" of a delayed peak and slow decline.

In uremia the renogram may indicate a dilated upper urinary tract and suggest the degree of renal function.

Several renogram tracings are illustrated in the article.—*John T. Underberg, M.D.*

LOKEN, MERLE K., STAAB, EDWARD V., VERNIER, ROBERT L., and KELLY, WILLIAM D. Radioisotope renogram in kidney transplants. *J. Nuclear Med.*, Oct., 1964, 5, 807-810. (From: Departments of Radiology, Pediatrics and Surgery, University of Minnesota Hospitals, Minneapolis, Minn.)

The authors describe the application of radioactive renography to follow the clinical course of patients who have received kidney transplants. Serial renograms are obtained immediately postoperatively, every 24 hours after surgery for 1 week and at various times thereafter. The report is based on 15 patients who were given varying doses of steroids and other immunosuppressive agents to prevent a rejection reaction of the renal transplant.

One brief case history including serial renography, blood and urine chemical determinations and immunosuppressive drug therapy schedule is presented.

It is to be noted that the renographic pattern does not always correlate with the clinical status, as abnormal patterns persist despite improvement in urine output and blood chemistries.

The most consistent pattern seen during a renal injection reaction is gradual depression of the functional peak and an excretory phase, either delayed and/or elevated.

A dose of 20 μ c of I^{131} hippuran is administered intravenously with the patient in the supine position. The probes are placed over the lower anterior abdominal-pelvic area as the kidneys are transplanted into the anterior iliac fossae. However, in smaller patients the probe position is more critical because of background activity from the urinary bladder.—*Richard Pfister, M.D.*

DE BERNARDI, E., and SPAZIANTE, G. Recovery of the renal function after surgical intervention. *Radiol. clin.*, 1964, 33, 217-222. (From: Istituto di Radiologia e Radiumterapia, Ospedali Riuniti "S. Giovanni di Dio e Ruggi D'Aragona," Salerno, Italy.)

In the course of pyelographic studies, the findings, at times, may seem such as to warrant nephrectomy.

The authors report 2 cases in which good recovery of renal function occurred after conservative therapy. One case had a large branched renal calculus and the other a polycystic kidney. They point out the value of renal arteriography in assessing the parenchymal lesions and place special emphasis on the late nephrogram. They also mention several other cases of good recovery in which the renal function was poor.

The patients, in whom nephrectomy appears the ultimate choice, may develop reversible changes.

It is felt that arteriography usually provides the answer as to whether less drastic surgery is indicated.—Robert E. Brubeck, M.D.

PALUBINSKAS, A. J., PERLOFF, DOROTHEE, and WYLIE, E. J. Curable hypertension due to renal artery lesions. *Radiol. clin.*, 1964, 33, 207-216. (From: Departments of Radiology, Medicine, and Surgery, University of California School of Medicine, San Francisco, California.)

The distinctive roentgenologic features and the results of corrective surgery for the major lesions of the renal artery responsible for secondary hypertension are described. A group of 167 hypertensive patients with renal artery lesions seen at the University of California Medical Center, San Francisco, since 1952, are used for the study. The types of renal artery lesions are divided into 5 groups.

The clinical and physical findings associated with most positive studies included: a recent onset of the hypertension with the elevation in blood pressure beginning at an age of less than 20 years or more than 50 years, increasing severity of the hypertension, malignant hypertension with papilledema, bruit over the epigastrium or flank, and evidence of atherosclerosis obliterans involving the abdominal aorta.

(1) *Atherosclerotic narrowing of the renal artery*, usually occurring at the orifice, was found in 77 patients. The male to female ratio was approximately 1.5:1, with a mean age of 57 years. The lesion was bilateral in about one-half of the patients. Usually, atheromatous plaques were also seen in the abdominal aorta and its major pelvic branches. Fifty-one of the patients had surgery. Endarterectomy was the most common procedure performed; however, nephrectomy or a splenorenal shunt was the elected procedure in a few patients. Of the 51 patients who underwent surgery, the blood pressure returned to normal in 18 per cent; 27 per cent had definite improvement; 27 per cent had no persistent lowering of their blood pressure; and 22 per cent died. Three patients had insufficient follow-up.

(2) Sixty-six cases of *fibromuscular hyperplasia* were seen and 86 per cent were women. Narrowing of the main renal artery due to fibromuscular hyper-

plasia usually occurred in the middle and distal thirds. The location and extent of the lesion determined the surgical procedure and the remediability. A corrective surgical procedure was performed on 38 of the 66 patients. The blood pressure became normal in 50 per cent of the 30 patients who had sufficient follow-up. The blood pressure decreased, but did not return to normal in 20 per cent. Surgery did not cause any significant improvement in 27 per cent.

(3) *Combined atherosclerosis and fibromuscular hyperplasia* was found in 7 patients. The atheromatous narrowing was found in the proximal portion of the renal artery, whereas the narrowing due to fibromuscular hyperplasia was found in the more distal portions. All were older women except one 27 year old male. Two cases were operated upon, but the results are not reported.

(4) Six patients had a *diminutive renal artery* without an isolated area of narrowing. There was an associated hypoplastic or atrophic kidney in these cases. Nephrectomy was performed in 2, with the blood pressure returning to normal in 1. No distinction could be made between primary and secondary reduction in size of the renal artery.

(5) The fifth group included 11 hypertensive patients with *miscellaneous renal artery lesions*. The lesions reported comprise: 2 cases of primary renal artery thrombosis; 1 of renal artery occlusion from multiple systemic emboli; 1 of diffuse intimal hyperplasia; 1 of intimal fibrosis; 4 of aneurysm of the renal artery unassociated with fibromuscular hyperplasia; 1 of arteriovenous fistula; and 1 of dissecting aneurysm of the aorta with involvement of the renal artery orifice.

The authors state that earlier renal arteriographic examination and the advances in reconstructive arterial procedures should increase the number of good operative results obtained in unexplained hypertension.—Everett E. Smith, M.D.

KAISER, THOMAS F., and FARROW, FRANKLIN C. Injury of the bladder and prostatomembranous urethra associated with fracture of the bony pelvis. *Surg., Gynec. & Obst.*, Jan., 1965, 120, 99-112. (From: Deaconess Hospital, Emergency Hospital, and Sisters of Charity Hospital, Buffalo, and Division of Urology, School of Medicine, State University of New York, Buffalo, N. Y.)

Lower urinary tract trauma resulting from pelvic fractures has become much more frequent with increased automobile travel, and while previously almost endemic to areas of heavy industry, this type of injury is now nationally dispersed.

Although the distended bladder can be ruptured by a sudden force, with or without fracture, the nondistended bladder and prostatomembranous urethra cannot be injured by a nonpenetrating force, unless the pelvic circle is broken. One of every 10

patients with disrupting fracture of the pelvis will have an associated rupture of the bladder or prostatomembranous urethra.

The great majority of bladder ruptures are extraperitoneal in type, and occur on the anterior or anterolateral surface near the bladder neck. Intrapertitoneal rupture is less frequent and may occur in distended bladders without the presence of fracture. The unequivocal diagnosis of extraperitoneal or intraperitoneal rupture is impossible from clinical examination alone.

Retrograde cystography is rapid, safe, and accurate. In extraperitoneal rupture the medium is seen to extravasate near the bladder neck, but may not be visualized if an inadequate amount or concentration of the contrast material has been injected. In intraperitoneal rupture the cystogram is typical, with the opaque material assuming an hourglass configuration with the partially filled bladder as the lower half and the medium in the pelvic peritoneal cavity as the upper half, and with contrast even outlining bowel loops.

Prostatomembranous urethral rupture may be diagnosed on rectal examination by the prostate being retracted out of its normal position and a boggy, fluctuant mass palpable in its place. Although a urethrogram is not necessary for diagnosis, urethrocystograms are recommended for confirmation as well as the possibility of bladder damage.

The "teardrop bladder" is a configuration where the bladder is compressed and lifted out of the pelvis by laceration of lateral pelvic vessels and muscles and subsequent hemorrhaging into the pelvic basin, and is not strictly a traumatic injury of the bladder. In this instance no surgery is indicated and the bladder returns to its normal position and contour spontaneously within a period of 2 weeks.

With bladder or prostatomembranous urethral rupture immediate operation is imperative. The detailed surgical management is outlined.—*Mark D. Reiss, M.D.*

BLOOD AND LYMPH SYSTEM

CHOI, JAE KEY, and WIEDEMER, HALL S. Chyluria: lymphangiographic study and review of literature. *J. Urol.*, Dec., 1964, 92, 723-727. (Address: Dr. Wiedemer, 335 Glassner Avenue, Mansfield, Ohio.)

Chyluria has been classified in two forms: the parasitic (tropical) and nonparasitic (nontropical), based on etiology. In the former the lymphatic blockage is most commonly caused by Filaria but other parasitic diseases have also been incriminated. Nonparasitic chyluria is less common and may be associated with trauma, infiltrating neoplasm, infection, or congenital defects. A few cases have been reported in association with tuberculous peritonitis, diabetes, and bronchial asthma. As a result of one or more of the etiologic factors, there is valvular in-

competency with dilatation of the normal lymphatic channels, resulting in a free flow of chyle from the intestinal tract to the urinary tract or in a reverse direction.

The authors report a patient believed to have the nontropical form of hematochyluria. This patient had intermittent severe chyluria over a period of 28 years. During the latter number of years of his illness, he had rather marked hematochyluria. His past history showed bronchial asthma but no exposure to tropical diseases. The lymphangiogram demonstrated marked telangiectasis of the lymphatic vessels in the paraaortic region and in the hypogastric and sacral groups. The femoral and inguinal lymph nodes were incompletely opacified and there was an anastomosis with the renal lymphatics as demonstrated by the combined lymphangiogram and excretory urogram. The site of the lymphatic block appeared to be the level of the thoracic duct.

The etiology, pathogenesis and treatment of chyluria are discussed, and the authors include 3 illustrations of the lymphangiograms.—*George W. Chamberlin, M.D.*

KISHIMOTO, TAKASHI, HIGUCHI, TERUO, ENDO, MINORI, and KAI, YOSHIO. Lymphography in a patient with unilateral chyluria. *J. Urol.*, Nov., 1964, 92, 574-578. (From: Department of Urology, Self-Defense Forces Central Hospital, Tokyo, Japan.)

This report includes a study of 2 patients with post-filaria chyluria or hematochyluria. In one patient a retrograde pyelogram demonstrated large dilated lymphatic vessels extending from the pericalyceal structures of the kidney into the retroperitoneal lymph nodes. An immediate lymphangiogram then showed the connection between the retroperitoneal lymphatic vessels and the renal lymphatic vessels. Following surgical removal of all of the dilated lymphatics in the renal hilus, the chyluria cleared and the subsequent retrograde pyelogram showed no evidence of a lymphatic anastomosis.

In a second case, the lymphatics extending from the retroperitoneal area to the kidney were demonstrated by lymphography.

In patients with chyluria due to filariasis, the principal lesions are found in the lymphatics of the pelvic, lumbar and retroperitoneal regions. In some instances, however, the entire lymphatic system including the thoracic duct is extensively involved. The lymphatic vessels lose elastic tissue as they dilate and the walls become thickened and the valves incompetent. Thickened walls, incompetent valves, and reactive as well as cicatricial changes develop, leading to obstruction of the afferent lymphatics. The degree of visualization may be quite variable, reflecting the extent of the underlying pathologic process.

Measurements to control chyluria include a low fat diet, bed rest, medication, and instillation or irrigation of the renal pelvis with a sclerosing agent and surgery. The surgical method of choice appears to be removal of all of the lymphatics around the renal vascular pedicle.

The authors include 3 illustrations of the lymphangiograms.—*George W. Chamberlin, M.D.*

SKELETAL SYSTEM

EECKELS, R., VINCENT, J., and SEYNHAEVE, V. Bone lesions due to smallpox. *Arch. Dis. Childhood*, Dec., 1964, 39, 591-597. (From: Departments of Paediatrics and Anatomy, Levanium University, Léopoldville, Congo Republic, Africa.)

The authors report 3 cases of osteomyelitis variolosa in which they had the opportunity of performing a histologic study of bone.

The patients varied in age from 2 years 2 months to 6 years. These children developed painful swellings of the arms and legs when the skin lesions were healing. Roentgenologic studies revealed strikingly symmetric involvement of the distal parts of the humeri, the radii, the ulnae and metacarpals, and the tibiae, fibulae, and metatarsals. The other bones were normal. There were severe, generalized periosteal reactions along the shafts of the involved bones as well as multiple areas of patchy destruction, especially around the wrists and ankles. Narrow bands of translucency underlay the cartilaginous plates of some of the metaphyses. Follow-up roentgenograms showed diminution of the periosteal reaction, overall densification of the whole bone and progression of the epiphyseal and metaphyseal destruction.

Histologic examination indicated the primary bone lesion to be a proliferating arteritis, leading to fibrosis, necrosis, and bone resorption. A bacterial etiology was thought to be unlikely.—*Arthur E. Childe, M.D.*

GORTVAI, PETER, and ROPER, ANTHONY. Pott's disease with congenital anomalies of the spine. *Brit. J. Surg.*, Nov., 1964, 51, 841-845. (From: Mpilo Central Hospital, Bulawayo, Southern Rhodesia.)

Tuberculous osteomyelitis of the spine is common in Africa. Two cases of unproven, but presumed tuberculous abscess associated with congenital spinal anomalies are reported.

The first case was a 14 year old African female with a gibbus of the lower cervical spine roentgenographically resembling a Klippel-Feil deformity. Paraplegia had recently occurred and the patient was hospitalized. Bed rest and antituberculous chemotherapy were ineffective; therefore, an anterolateral decompression of the lower cervical cord was done with subsequent recovery of the patient. The early operative approach to the treatment of paraparesis in such a case is advocated.

The second case was a 22 year old African male with a spinal deformity and cold paravertebral abscess which was drained and treated with antituberculous chemotherapy. A draining sinus developed which yielded *Staphylococcus aureus*. Roentgenograms revealed a 90° mid dorsal kyphoscoliosis with a large paravertebral abscess and two pathologic rib fractures. Since chemotherapy was ineffective, an anterolateral decompression was performed, a 300 ml. cavity drained and a sequestrum having the appearance of a hemivertebra removed. The patient recovered. The hemivertebra and rotation of the spine indicated that the spinal deformity was congenital in origin.

Spinal deformity and paraplegia should suggest the diagnosis of Pott's disease. Tuberculosis of the spine characteristically produces kyphosis, and lateral angulation may also be present; however, there is neither true lateral curvature or rotation of the spine. In advanced disease the adult spine tends to become stabilized by the development of a bony anterior fusion.

The etiologic agent in chronic spinal infections may, more rarely, be *Staphylococcus pyogenes*, *Escherichia coli*, or *Brucella melitensis*.—*John T. Underberg, M.D.*

DOWNS, W. J. Multiple pseudocystic tuberculosis of bone. *Canad. M.A.J.*, Dec. 12, 1964, 91, 1275-1277. (From: Aberhart Memorial Sanatorium, Edmonton, Alberta, Canada.)

The author reports a case of pseudocystic tuberculosis of bone in a 32 year old woman with multiple rounded, or oval, translucencies of skull, ribs, right clavicle and ilium. Both of the eleventh rib abscesses were proved to be due to *M. tuberculosis* bacteriologically. Gastric lavage was also positive, although there was no gross pulmonary disease. Guinea-pig inoculations were similarly positive.

Under treatment for tuberculosis there was marked improvement clinically but the roentgenologic findings did not change.—*Peter C. Truog, M.D.*



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POSTIRRADIATION SIALADENITIS

A STUDY OF THE CLINICAL FEATURES, HISTOPATHOLOGIC
CHANGES AND SERUM ENZYME VARIATIONS FOLLOWING
IRRADIATION OF HUMAN SALIVARY GLANDS

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BETHESDA, MARYLAND

A SYNDROME of acute swelling, tenderness and pain which subsides within a few days has been long recognized as a sequel of irradiation of the major salivary glands. In 1911 Bergonie and Speder³ described 10 instances of salivary gland swelling occurring a few hours after radiation therapy. Ceresole's⁷ report of 1912 remains one of the most complete descriptions of the postirradiation reaction of the salivary glands. He noted that 21 of 84 patients receiving radiation to the head and neck regions had salivary gland tumefaction which began 5 to 6 hours after irradiation, reached a maximum at 8 to 12 hours, remained another 5 to 6 hours, and then disappeared rapidly. Desjardins's⁸ observation that "... With the exception of Ceresole, . . . , nearly all those who have mentioned the subject have done so only incidentally and have failed to distinguish the early from the late reactions . . . " is as applicable today as it was in 1931.

Studies of the reaction of the salivary glands of animals to irradiation have been confined largely to histologic observations. Although the late histologic sequelae of human salivary glands exposed to radiation have been recorded,^{10,23} descriptions of acute alterations are uncommon.

Clinical radiotherapy studies¹ on patients with malignant neoplasms of the naso- and oro-pharynx have provided a unique opportunity to observe and study the short term effects of radiation on salivary gland tissue.

METHODS

PATIENTS

The patients studied had malignant neoplasms of the naso-, hypo-, or oro-pharynx. The clinical descriptions and the serum chemical changes to be considered were made on the 33 patients listed in Table 1. There were 4 categories of patients. Eighteen were patients participating in a

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time-dose radiation study from July, 1960 to July, 1963. These patients received 1,500 to 2,750 r in a single treatment session. Five were from a group of patients who received large single doses of radiation 24 hours prior to surgery. Five patients, who were hospitalized at the National Naval Medical

Center (NNMC), were selected for study because they received tumor irradiation in the dose range of conventional therapy. The remaining 5 patients were seen on a consultative basis by the Radiation Therapy Service and were irradiated according to the dictates of the clinical situation.

TABLE I

PATIENTS, RADIATION FACTORS AND SERUM AMYLASE CHANGES FOLLOWING IRRADIATION

Patient, Unit No.	Age/Sex	Diagnosis†	Radiation Therapy			Serum Amylase (Somogyi Units)	
			Tumor Dose r	Salivary Gland Mass Exposed		Pre-irra- diation	Maximum Post- irradiation Response
				<50%	>50%		
JA 04-05-07	58/M	EC, pharynx	2,000	x	x	125	1,926
BB 04-77-34	31/F	EC, tongue	1,000		x	120	1,660
MB 03-17-20	42/F	EC, nasopharynx‡	2,500		x	(113)	481
RB 03-63-41	48/M	Ly, tonsil	1,500		x	126	1,800
HC 04-11-32	48/M	EC, pharynx	2,500	x		116	444
HC 03-72-85	71/M	EC, soft palate	2,500		x	(113)	1,625
WD 00-01-20	74/M	EC, cheek§	100	x		283	1,250
GD 03-94-60	53/M	Ly, palate	1,500	x		113	746
HF 03-38-66	56/M	EC, pharynx	2,500		x	(113)	2,840
EF 04-03-16	42/F	EC, tongue	2,500		x	145	1,510
DG 04-19-94	40/F	Le, tongue	300		x	80	1,923
BG 03-73-93	17/M	EC, tonsil	2,000		x	59	1,840
CG 03-36-11	55/F	EC, tongue	2,500		x	72	2,230
JH 04-48-78	43/F	EC, pharynx	2,000		x	243	5,800
CH 04-09-11	63/M	UC, parotid	1,000		x	(113)	842
EJ 04-17-98	59/M	EC, piriform sinus§	2,500		x	277	2,080
GK 01-49-24	62/F	EC, soft palate	1,000		x	105	1,260
MK 02-96-21	67/F	MEC, minor salivary§	1,000		x	112	2,630
GM 03-48-86	56/M	EC, larynx	2,500	x		68	181
SM 04-80-38	74/F	EC, buccal mucosa	200		x	124	260
AN 04-35-51	47/M	EC, buccal mucosa	2,000		x	85	1,180
RP 04-43-89	52/F	EC, gingiva	1,000		x	(113)	9,300
HP 04-46-39	64/M	EC, piriform sinus	400	x		198	1,140
RP 04-81-95	58/M	EC, larynx	1,000	x		77	1,760
ER 04-91-75	60/F	EC, tongue	2,000		x	69	1,320
RS 04-09-09	53/M	EC, palate	2,500		x	80	5,245
WV 03-65-56	53/M	EC, floor of mouth	2,750		x	110	1,600
LW 01-40-05	57/M	EC, epiglottis	2,500	x		69	260
LD 124-63**	47/F	EC, tongue	100		x	135	211
EL 36-63**	25/M	Lymphoma	300*		x	67	473
JL 106-63**	52/F	EC, pharynx	150		x	106	386
LL 103-63**	60/M	Melanoma	100*	x		(113)	159
CM 114-63**	54/M	Ad, parotid	150*	x		149	190

* Cobalt 60 source.

† EC—epidermoid carcinoma.

Ly—lymphoma.

Le—leukemia.

‡ Treated 3,700 r 4 years earlier.

§ Previous excision.

|| Previous laryngectomy.

** NNMC patients.

UC—undifferentiated carcinoma.

Ad—adenocarcinoma.

MEC—mucoepidermoid carcinoma.

The histopathologic observations were made on 22 consecutive patients participating in a presurgical irradiation study. It should be noted that only 5 of these patients are included in the previously described group although the clinical features were evident in each irradiated patient. Of this group, 16 patients received 1,000 to 2,000 r in a single treatment session, 24 hours prior to having radical excision of the tumor and lymph node bearing tissues. Six patients of this group had similar surgical therapy but were sham irradiated 24 hours prior to surgery.

RADIATION

All but 2 patients who received radiation therapy were treated by a Van de Graaff Generator operating at 2.0 mev. (half value layer 12 mm. Cu at a focal-skin distance of 110 cm.), delivering a constant exposure rate of 75 r per minute. All patients received a single exposure of radiation while being studied and fractionated therapy was not used except in those few additional cases in which the fractionation of the dose is made clear in the text. The patients were treated while seated in a Franklin rotating chair, which was stationary in all instances. The patient's head was immobilized and the treatment field was continuously monitored by television control of the transmitted beam.²

Two patients from the National Naval Medical Center were treated by radiation from a cobalt 60 source (half value layer 11 mm. Pb, focal-skin distance 55 cm., dose rate 36 r per min.). The radiation characteristics are judged to be sufficiently similar to those of the 2.0 mev. Van de Graaff Generator to allow inclusion of these patients in this study.

CHEMICAL EVALUATION OF SERUM

The sera, except for the 3, 6, 9 and 12 hours postirradiation samples, were all obtained with the patient in the fasting state in the morning. Serum amylase, lipase, SGOT, SGPT and alkaline phosphatase were determined by the chemistry

section of the Department of Clinical Pathology at the National Institutes of Health. Lactic acid dehydrogenase (LDH) was determined by the authors according to the Berger-Broida modification of the method of Wroblewski.¹⁹ The serum amylase method used is that of Van Loon *et al.*²²

HISTOPATHOLOGY

Tissues from all 22 patients in a double blind preoperative radiation study group were evaluated. The surgical specimen included the submaxillary gland and the lower pole of the parotid gland in most cases. The sublingual and minor salivary glands also were available in some specimens.

The surgical specimens were fixed in formaldehyde and sections stained with hematoxylin and eosin. The 22 cases were evaluated as a group.

SIALOGRAPHY

Parotid sialography was done by the cannulation of Stensen's orifice with a plastic catheter and injecting hypaque solution until appearance of the characteristic parotideal pain; 1.0 to 1.5 cc. was the usual amount injected. Roentgenograms were taken with the catheter in place and, following preliminary inspection of these films, the catheters were withdrawn and the patient was allowed to draw upon a freshly cut lemon. Repeat roentgenograms were taken at 5 minutes and 24 hours after this stimulation to check for evacuation of the contrast medium.

RESULTS

1. CLINICAL OBSERVATIONS

The earliest and an invariable complaint by the patients was that of dryness of the mouth. It usually occurred between 2 and 6 hours after irradiation. In patients receiving less than 1,000 r this complaint was elicited by interrogation, although its appearance was often delayed beyond 6 hours.

Parotideal pain and tenderness appeared at 4 to 6 hours after irradiation. Noticeable enlargement of the parotid and submaxil-

lary glands occurred simultaneously and often was of gigantic proportions. This swelling appeared to reach its maximum between the twelfth and twenty-fourth hours after irradiation and then rapidly subsided. Patients complained variously of tingling or burning sensations over the glands and of tightness of the facial skin. The latter complaint was common in patients with the greatest degree of swelling.

Physical examination disclosed exquisite tenderness to palpation over the swollen salivary glands. This was regularly most marked in the infra-auricular portion of the parotid gland and frequently was present within the first hour after irradiation. The submaxillary gland enlargement was often of impressive proportions and the glands were tender to palpation. The glands were firm and without discernible edema. In many instances the mandibular angle was obscured by the swelling and, in almost all respects, these patients mimicked the textbook picture of epidemic parotitis.

Temperature elevation was rare. Five of the 28 patients had peak temperatures in excess of 38° C. during the 24 hours after irradiation. Two of these were being treated for infections and had been having temperature elevations in this range before the radiation exposure. In 8 patients a peripheral white blood cell count was performed at 24 hours after irradiation. Only one had a leukocytosis (11,200); this, however, is one of the previously mentioned patients who was receiving treatment for a systemic infection and had a peripheral white blood cell count of 10,000 immediately prior to irradiation.

The postirradiation stomatitis, commonly known as mucositis by radiotherapists, is a regular sequel to radiation of the oral cavity. Edematous mucositis was noticeable within the first day in the present group of patients and the other changes consisting of erythema and the characteristic epithelial patches appeared by the end of the first week.

It is emphasized that, despite the dramatic appearance and severity of the clinical

reaction, simple symptomatic care alone was sufficient to provide comfort for these patients. By the third day the salivary gland reaction, as manifested by swelling, was largely gone although tenderness to palpation persisted for as long as 7 days in some cases.

2. SERUM CHEMICAL OBSERVATIONS

Amylase. The patients, radiation factors and serum amylase values are listed in Table 1. Baseline values of serum amylase were obtained in 23 patients (56 separate determinations). The mean values for amylase ranged between 68 units (G.M., 03-48-86) and 283 units (W.D., 00-01-20). An operation entering the parotid gland had been performed 14 days prior to the determination in the latter patient and may have played a role in his high serum amylase value. Patient H.P. (04-46-39) and E.J. (04-17-98) were recuperating from radical neck dissection operations which may have contributed to their elevated serum amylase levels. There is no suggestion as to the cause of the pre-irradiation hyperamylasemia in J.H. (04-48-78).

The arithmetic mean of serum amylase values of the entire group, prior to irradiation, was 124 ± 14 Somogyi units (S.E.); the geometric mean was 113 Somogyi units. Values of both are well within the limits of normal (60-160 Somogyi units).

The geometric means of the serum amylase values after irradiation are represented graphically (Fig. 1) and describe a marked and transient elevation. A sharp rise is clearly evident by the third hour after irradiation. The peak elevation occurred between the ninth and thirty-sixth hours, at which times the mean serum amylase values ranged between 9 to 18 times the pre-irradiation values. The peak serum amylase was 9,300 Somogyi units observed in patient R.P. (04-43-89) at 9 hours after irradiation. The serum amylase receded sharply after the second postirradiation day and values in the normal range were the rule by and after the fourth day.

All enzyme analyses subsequent to the

fifth postirradiation day have been grouped together. Most of these samples were obtained at the time of a follow-up examination in the outpatient department. In all, there were 38 separate determinations on 16 patients. The arithmetic mean serum amylase in this follow-up period was 99 ± 18 Somogyi units. The geometric mean was 83 Somogyi units. Again, as was the case with the pre-irradiation determinations, these values are within the normal range. Application of Student's *t* test failed to demonstrate any significant difference between the pre- and postirradiation means ($t = 0.195$, $df = 41$).

To allow further analysis of the data, each patient's serum amylase response was expressed in terms of a log ratio,

$$\log \frac{\text{peak postirradiation serum amylase}}{\text{mean pre-irradiation serum amylase}}$$

(hereafter referred to as the geometric ratio). Each patient, thus, served as his own "control." In the 6 cases in which

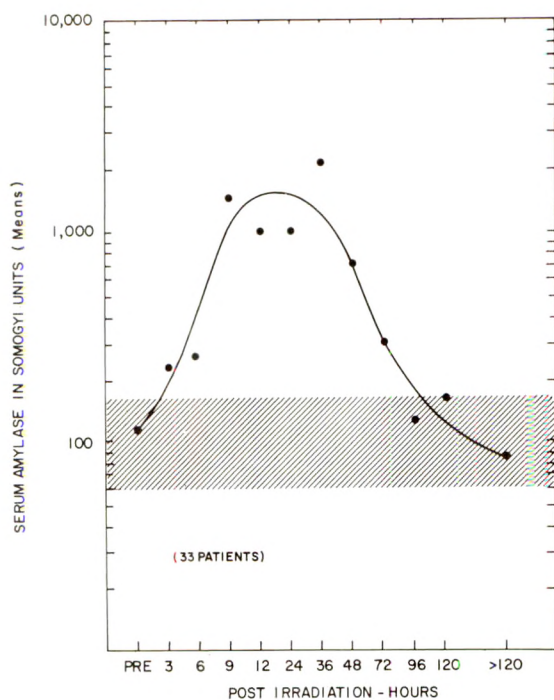


FIG. 1. Serum amylase response following irradiation. Hatched area indicates the normal range of serum amylase.

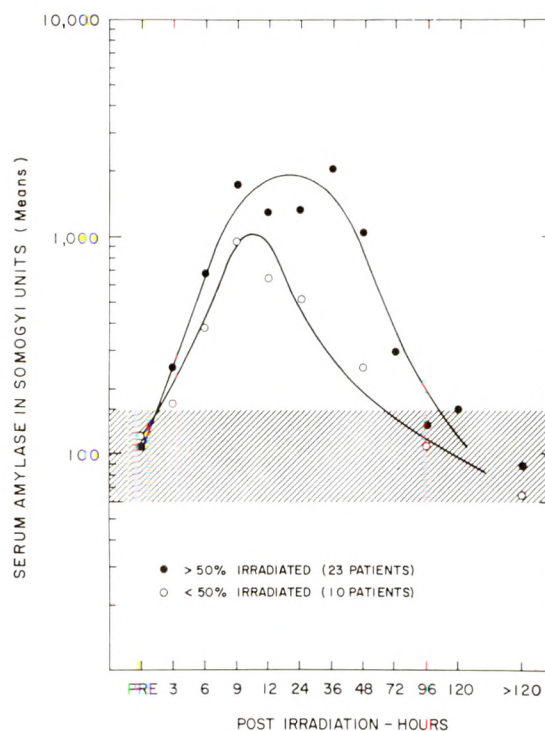


FIG. 2. Serum amylase response as related to salivary gland mass irradiated.

pre-irradiation sera were not available, the over-all pre-irradiation mean 113 was used. The ratios transformed to log values appear to be distributed normally.

Amylase Response As Related to Volume of Salivary Mass Irradiated. From the therapy records 10 patients were judged to have had less than half of the gland mass irradiated; 23 patients had more than half of the gland mass irradiated (Table II). The means of the geometric ratio calculated to 0.660 in the former group and 1.131 in the latter. By the *t* test the difference was highly significant ($p < 0.005$), indicating that the magnitude of the serum amylase response is directly related to the volume of salivary mass irradiated. This finding is illustrated in Figure 2.

Amylase Response As Related to Radiation Dose. Nine patients (4 NIH, 5 NNMC) received less than 1,000 r, 8 patients received between 1,000 and 2,000 r and 16 patients received more than 2,000 r, in all instances in a single dose.

TABLE II
MEAN SERUM AMYLASE RESPONSE (IN SOMOGYI UNITS) FOLLOWING IRRADIATION

	Pre-irradiation	Hours after Irradiation										
		3	6	9	12	24	36	48	72	96	120	>120
Total Group (33 patients)												
Geometric Mean	113	220	232	1,407	990	986	2,054	680	292	123	158	83
± Standard Error	(104-122)	(182-266)	(194-277)	(973-2,043)	(779-1,257)	(809-1,200)	(1,736-2,432)	(532-870)	(233-365)	(110-137)	(111-224)	(72-96)
Determinations	27	19	24	9	19	30	4	14	8	6	2	16
Less Than 50% Salivary Mass Irradiated (10 patients)												
Geometric Mean	120	170	376	944	634	501	—	244	—	107	—	64
Determinations	9	6	8	3	7	9	—	4	—	2	—	3
More Than 50% Salivary Mass Irradiated (23 patients)												
Geometric Mean	109	248	666	1,718	1,280	1,320	2,054	1,026	292	134	158	88
Determinations	18	13	16	6	12	21	4	10	8	4	2	13
Less Than 1,000 r (9 patients)												
Geometric Mean	130	306	311	601	474	403	—	315	—	—	—	214
Determinations	8	3	8	3	7	9	—	1	—	—	—	1
More Than 1,000 r (24 patients)												
Geometric Mean	106	207	732	2,150	1,521	1,445	2,054	722	292	123	158	78
Determinations	19	16	16	6	12	21	4	13	8	6	2	15

TABLE III
SERUM ENZYME ACTIVITIES BEFORE AND AFTER IRRADIATION

	Pre-irradiation†	Hours after Irradiation†										
		3	6	9	12	24	36	48	72	96	120	>120
Lipase (less than* 1.5 units)	1.18	1.34	1.27	1.15	1.13	0.75	0.51	1.02	0.86	0.33	1.7	0.78
Determinations	12	12	12	8	11	15	1	3	3	1	1	5
Alkaline Phosphatase (4-13 K.A. units)	7.33	6.4	6.6	6.6	6.4	7.16	—	6.0	—	—	—	10.2
Determinations	6	5	5	5	5	6	—	1	—	—	—	4
Lactic Acid Dehydrogenase (100-550 B—B units)	497	505	488	346	346	430	—	542	521	395	388	318
Determinations	12	11	12	8	8	11	—	4	4	2	2	3
SGOT (10-33 units)	20.6	13.4	16.7	16.0	15.3	17.9	14.0	18.2	33.0	19.2	18.5	15.6
Determinations	13	7	6	5	6	10	4	8	2	4	2	10
SGPT (10-33 units)	18.7	15.5	18.3	15.2	18.3	16.3	15.3	14.8	—	—	39	13.7
Determinations	8	7	6	5	6	9	3	4	—	—	1	6

* Normal values are indicated in parentheses.

† Mean values.

Using the geometric ratio for each group, the mean values were 0.545, 1.200 and 1.112, respectively, for the less than 1,000 r group, the 1,000-2,000 r group and the greater than 2,000 r group. The t test failed to show any significant difference among these groups.

When, however, the mean of the geometric ratios for the patients receiving less than 1,000 r (0.550) and the mean for the patients receiving more than 1,000 r (1.153) are compared, the difference is highly significant ($t=0.575$, $df=22$). This relationship is depicted in Figure 3.

Amylase Response As Related to Sex. There were 20 males and 13 females in this series. No sex difference in amylase response was evident from an analysis of the data ($t=0.575$, $df=31$).

Other Enzymes. Serial serum samples were analyzed for lipase, SGOT, SGPT, alkaline phosphatase and lactic acid dehydrogenase levels. Although some minor fluctuations were observed, neither a con-

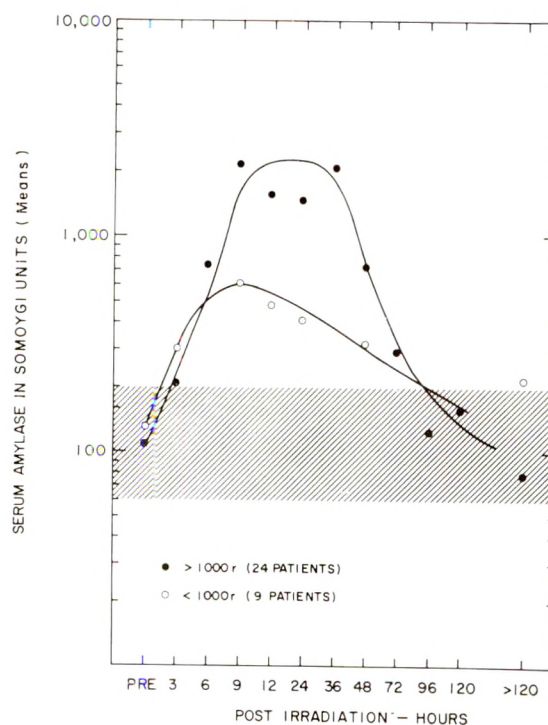


FIG. 3. Serum amylase response as related to radiation dose.

sistent pattern nor change of any magnitude was evident. A tabular summary of the data is presented in Table III.

3. HISTOPATHOLOGY OF IRRADIATED SALIVARY GLANDS

Table IV shows the patients, the primary pathologic diagnoses, the tumor radiation doses and the degree of acute inflammation and degeneration in the salivary glands. A single notation of the degree of change is made because the acute inflammatory changes closely paralleled degenerative changes.

No gross abnormalities of the salivary glands were recorded by the examining

pathologist. When salivary glands were involved with tumor, portions distant from the site of involvement were evaluated for acute postirradiation change.

Parotid Glands. Fifteen parotid glands were examined; of these 10 were irradiated. Without exception, parotid glands from irradiated patients exhibited acute inflammatory and degenerative changes. The reaction within a single gland was fairly uniform. There was greater variation in the intensity of reaction in glands from different patients.

(a) Inflammatory cell infiltrates. All irradiated glands were infiltrated by poly-

TABLE IV
REACTION OF SALIVARY GLAND TISSUE TO IRRADIATION

No.	Patient's Unit No.	Age	Sex	Diagnosis† and Primary Site of Tumor	Tumor Dose (r)	Acute Inflammation and Degeneration of Salivary Gland Tissue‡		
						Parotid	Submaxillary	Minor Salivary Glands
1	03-26-84	55	M	EC, larynx	2,000	NE§	+++	++
2	03-31-64	83	M	EC, buccal mucosa	2,000	NE	+++	++
3	03-28-54	48	F	EC, tongue and mouth	2,000	++++	++++	+++
4	03-40-32	24	M	MEC, hard palate	1,500	NE	NE	++
5	03-66-25	59	M	EC, larynx	1,000	NE	NE	+
6	03-38-43	72	M	EC, floor of mouth	1,000	++++	+++	++
7	02-99-87	43	M	MT, lower lip	1,000	+++	+++	++
8	04-43-89*	52	F	EC, gingiva	1,000	+++	++	++
9	04-09-11*	62	M	UC, parotid gland	1,000	+++	+++	NE
10	03-44-54	72	F	EC, alveolar ridge	1,000	NE	+++	++
11	04-03-02	52	F	EC, buccal mucosa	1,000	+++	+++	+
12	04-15-31	63	M	EC, floor of mouth	1,000	Left ++ Right NE	++ ++	++ —
13	04-77-34*	31	F	EC, tongue	1,000	NE	++	++
14	01-49-24*	61	F	EC, soft palate	1,000	+++	o	++
15	04-81-95*	58	M	EC, larynx	1,000	+++	+++	NE
16	04-95-36	57	M	EC, tonsillar pillar	1,000	++	++	++
17	04-27-11	62	F	MT, palate	sham	o	o	o
18	03-08-15	70	M	EC, base of tongue	sham	o	o	o
19	03-69-34	44	F	EC, larynx	sham	NE	o	o
20	04-11-72	53	M	EC, tongue	sham	o	o	o
21	04-46-39	63	M	EC, piriform sinus	sham	o	o	o
22	04-92-52	46	M	EC, tongue	sham	o	o	o

* Patients on whom serum amylase was studied.

† EC—epidermoid carcinoma.

MEC—mucoepidermoid carcinoma.

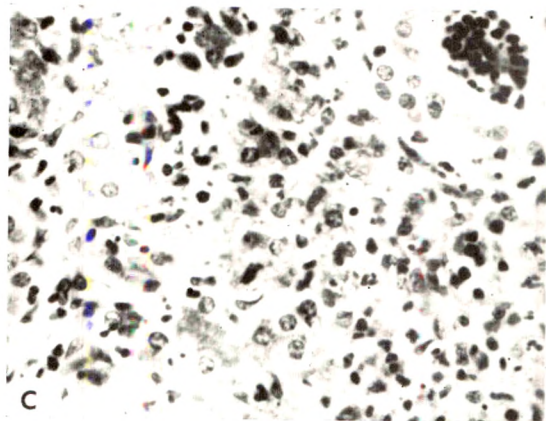
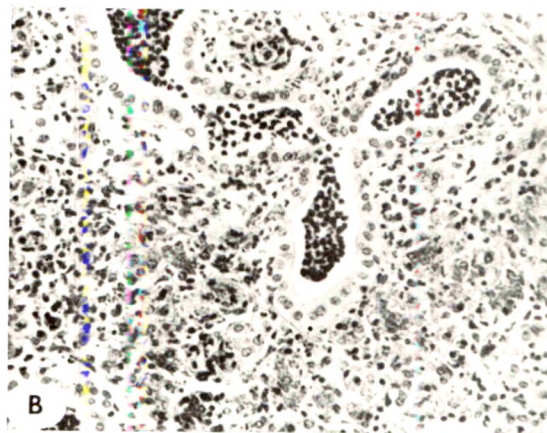
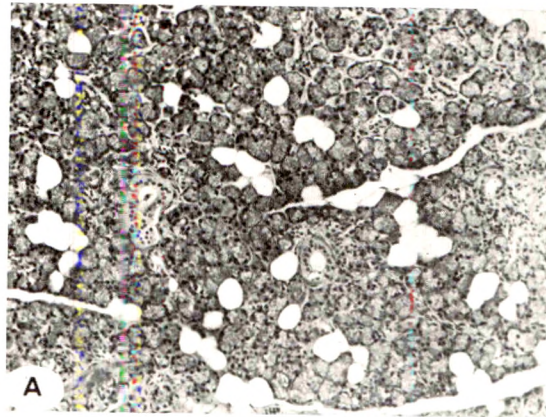
MT—mixed tumor.

UC—undifferentiated carcinoma.

‡ Graded response: o—no acute reaction; +++—acute inflammation with focal necrosis.

§ NE—not examined.

FIG. 4. Parotid gland tissue. (A) Normal, $\times 120$. (B) Tissue from patient (G.K., 01-49-24) 24 hours after receiving 1,000 r of radiation in a field including the parotid gland, $\times 210$. (C) Higher magnification of B demonstrates vacuolization of acinar cells, pyknosis of acinar cell nuclei and inflammatory cell infiltration, $\times 640$.



polymorphonuclear leukocytes, eosinophilic leukocytes and a few plasma cells. These cells tended to collect along interlobular septa where in some glands there was frank zonal suppuration. Inflammatory cells also tended to outline acini and some were seen among the granules in degenerating serous cells. A constant finding was the purulent exudate in the lumens of intercalated, interlobular and excretory ducts (Fig. 4, B and C) which appeared to be made up of degenerating polymorphonuclear leukocytes and degenerating acinar cells.

(b) Acini. In many glands the discrete outlines of acini were barely discernible and the serous cells had large irregular basilar vacuoles or spaces seeming to rupture cells and cleave them from the basement membrane (Fig. 4C). Many serous cells in an inflamed gland had pyknotic nuclei. Degenerative epithelial cells were part of the exudate in the lumens of ducts. Zymogen granules in some glands were pooled in acini and appeared free of cytoplasmic membrane envelopment. In other glands there was a marked reduction in the number of these granules.

(c) Ducts. In general, intercalated and intralobular ducts were dilated but the epithelial lining remained intact preserving the duct outline. The larger excretory ducts were only moderately dilated and contained less of the exudate than smaller ducts. There was no evidence of excretory duct obstruction in the sections examined.

The 5 parotid glands from sham irradiated patients revealed normal histologic features.

Submaxillary Glands. All but one of the

14 irradiated submaxillary glands showed acute inflammation and degenerative changes similar to those described in the irradiated parotid glands. There were purulent exudates in ducts, suppurative foci along interlobular septa, inflammatory cell infiltrates in serous acini, basilar vacuoles

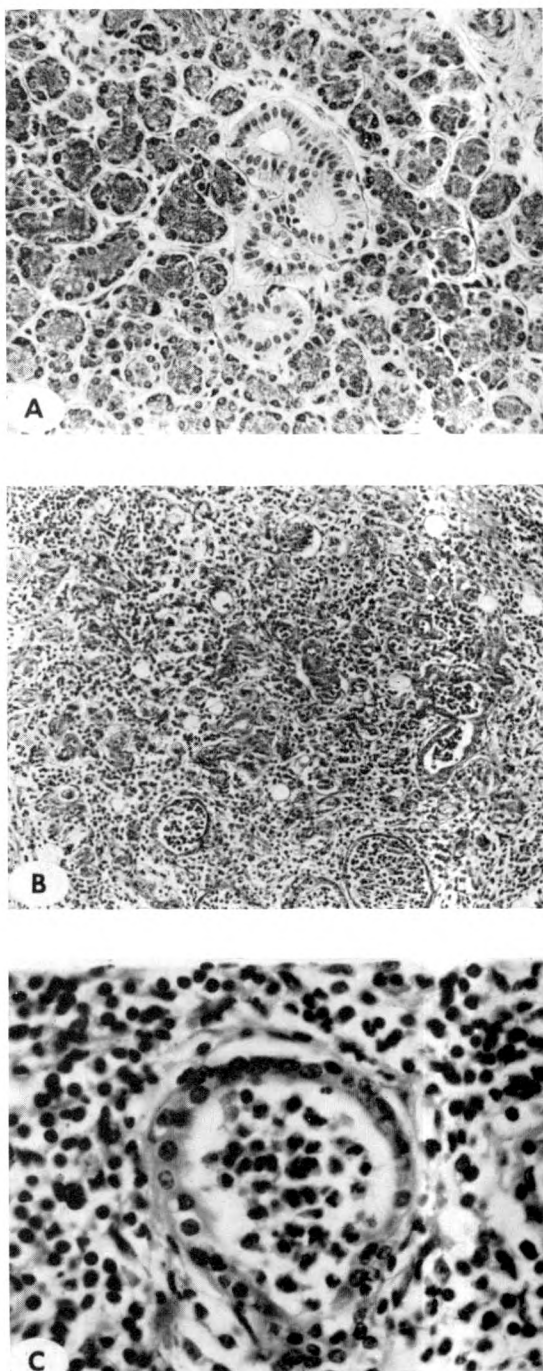


FIG. 5. Submaxillary gland tissue. (A) Normal structures with no discernible alteration in same patient as shown in Figure 4, B and C, whose submaxillary gland was not in the treatment field, $\times 210$. (B) Marked reaction (A.G., 03-28-54) 24 hours after treatment with 2,000 r. Serous cells are disrupted, acini are disorganized, the tissue is infiltrated by inflammatory cells but duct outlines are intact, $\times 120$. (C) Same as B, $\times 640$.

in serous cells, disrupted serous cells and pools of zymogen granules in acini (Fig. 5, B and C; and 6A). A marked degranulation of serous cells was noted in many of the glands. These reactions were more pronounced in patients receiving the larger doses of radiation and the density of inflammatory cell infiltrates closely paralleled the severity of degenerative changes in serous cells. In sharp contrast little, if any, discernible microscopic change was present in mucous cells (Fig. 6, A, B and C).

One submaxillary gland (G.K., 01-49-24) exhibited no acute change, yet there was a typical acute inflammation and degeneration in the parotid gland from the same surgical specimen (Compare 4B and 5A). The radiation field had been directed to a lesion of the soft palate so that the parotid gland was irradiated but not the submaxillary gland.

In the 6 submaxillary glands from sham irradiated patients, there were no acute changes.

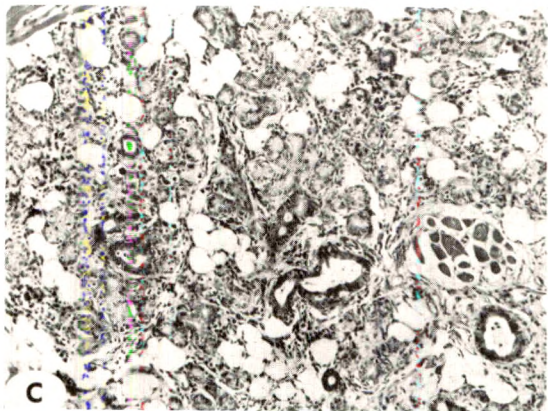
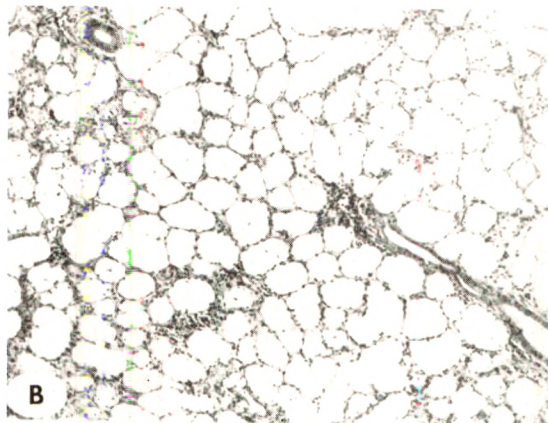
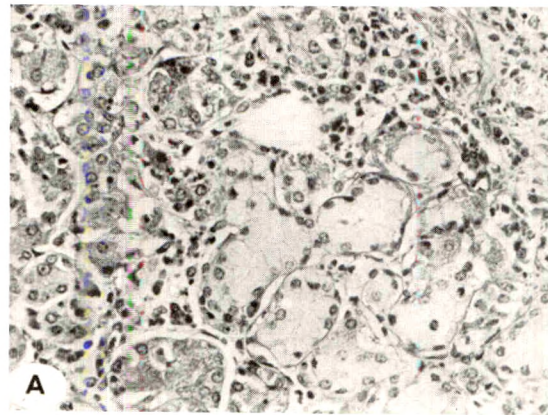
Sublingual and Minor Salivary Glands. The sublingual gland was present in only a few of the surgical specimens. Care was taken to discriminate between those changes due to duct obstruction or proximity of tumor and those changes due to radiation. The reaction in the sublingual and minor salivary glands varied with the composition of the glands. In those portions composed entirely of mucous cells, the acute inflammatory cell infiltrates were scant (Fig. 6B). In mixed glands acute inflammation and degeneration were present in serous acini and some exudate was present in small ducts (Fig. 6C).

Other Tissues. No attempt was made to evaluate tissues other than the salivary glands. The tissues surrounding the parotid and submaxillary glands were, in general, free from inflammatory cell infiltration. There was no evidence of vascular obstruction. Some acute inflammation and degeneration were present in lymph nodes.

Summary of Histopathologic Findings. The parotid and submaxillary salivary gland tissue of patients who received a



FIG. 6. (A) Submaxillary gland tissue from patient (M.H., 03-40-32) 24 hours after treatment with 1,500 r tumor dose. Serous acini contain vacuolated cells with pyknotic nuclei. Inflammatory cells are associated with degenerating serous acini, whereas mucous acini reveal little or no alteration, $\times 315$. (B) Sublingual salivary gland tissue from patient (J.V., 03-38-43) 24 hours after treatment with 1,000 r. Mucous cells are unaltered. Inflammation is present in foci containing a few serous acini, $\times 92$. (C) Lingual salivary gland tissue from same patient as in B. Inflammation and acute cell degeneration are present in serous cells; mucous cells are unaltered, $\times 92$.



single therapeutic dose of radiation 24 hours prior to surgery displayed an acute inflammatory response which was associated with marked degenerative changes in serous cells, and, in some cases, with foci of necrosis in the gland parenchyma. Mucous portions of the salivary glands revealed little, if any, histopathologic change. Glands from sham irradiated patients had no acute alterations.

4. OTHER OBSERVATIONS

Refractoriness of Salivary Glands to Radiation. H.P. (04-46-39), a patient with epidermoid carcinoma of the piriform sinus who had previously undergone a radical laryngopharyngectomy and right radical neck dissection, was referred for treatment of persistent tumor in the neck and retropharynx. A course of conventional radiation treatment, using the previously described technique, at a daily exposure rate of 400 r was initiated. The promptness and the degree of the postirradiation hyperamylasemia following the initial treatment were in all respects identical with those observed in other patients (Fig. 7).

Twelve calendar days (8 treatment days) later, by which time he had received 3,200 r, a repeat serial serum amylase study was made (Fig. 7). The patient had had 2 days without treatment (because of an intervening weekend) and the pre-irradiation serum amylase level was normal. Following the

400 r tumor dose on this occasion, the serum amylase level showed only a mild elevation although the port and radiation factors were identical with those used in the initial treatment.

Response to a Low Level of Radiation and a Small Field. Patient W.D. (00-01-20) had an epidermoid carcinoma of the skin of the

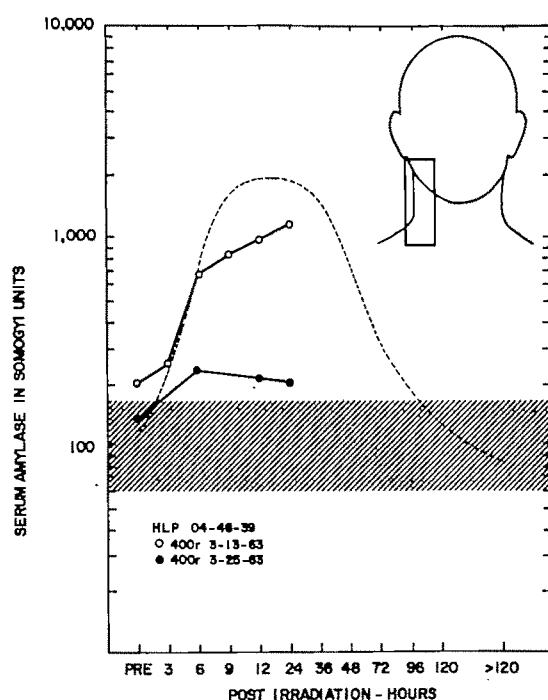


FIG. 7. Demonstration of a depressed serum amylase response following repeat irradiation. Open circles depict the serum amylase response to the initial 400 r. Closed circles depict the serum amylase response to 400 r 12 days later by which time 3,200 r had been delivered by fractionated therapy via the treatment field indicated.

cheek and was treated by conventional fractionated therapy. In order to evaluate the serum amylase response to a low dose and small port, the initial treatment was 100 r via a tangential field 10×4 cm. (Fig. 8). It is estimated that less than 25 per cent of the total salivary mass was irradiated. Despite this low dose, a serum amylase rise to 1,200 units was observed by the twelfth postirradiation hour, a four-fold rise from his baseline value.

Sialography. Parotid sialography was performed on one patient (J.H., 04-48-78) on the day prior to irradiation, 1 day, and at 20 weeks after irradiation. The first (pre-irradiation) examination was normal in all respects. Figure 9, A-D demonstrates good filling of the principal duct systems and the characteristic reticular pattern extending into the terminal acini. The posteroanterior study demonstrates normal gland size, and

again, the normal filling pattern. Evacuation was prompt and complete in the roentgenogram taken 5 minutes after stimulation.

In the immediate (1 day) postirradiation examination, the patient was able to tolerate less contrast medium (before parotid pain appeared) than on the prior examination. The postinjection roentgenograms (Fig. 10, A-D) show an enlarged gland, less duct filling and absence of the previously noted reticular acinar pattern. There is no duct dilatation to suggest duct obstruction. Another feature is the incomplete evacuation of the contrast medium 5 minutes after the removal of the catheter and stimulation (Fig. 10C). Moreover, radiopaque remnants were present within the gland on the next day and even on the twenty-fifth day following this examination (Fig. 10D).

The third sialography (20 weeks post-irradiation) showed a less detailed pattern

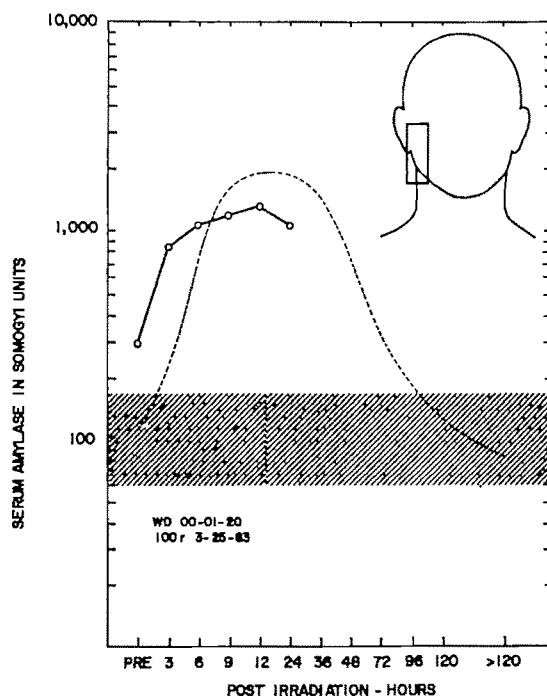


FIG. 8. Demonstration of the marked sensitivity of the salivary gland to irradiation. Open circles depict the serum amylase response following 100 r delivered via the treatment field (25 per cent of salivary gland mass exposed). Broken line is the over-all average (see Fig. 1).

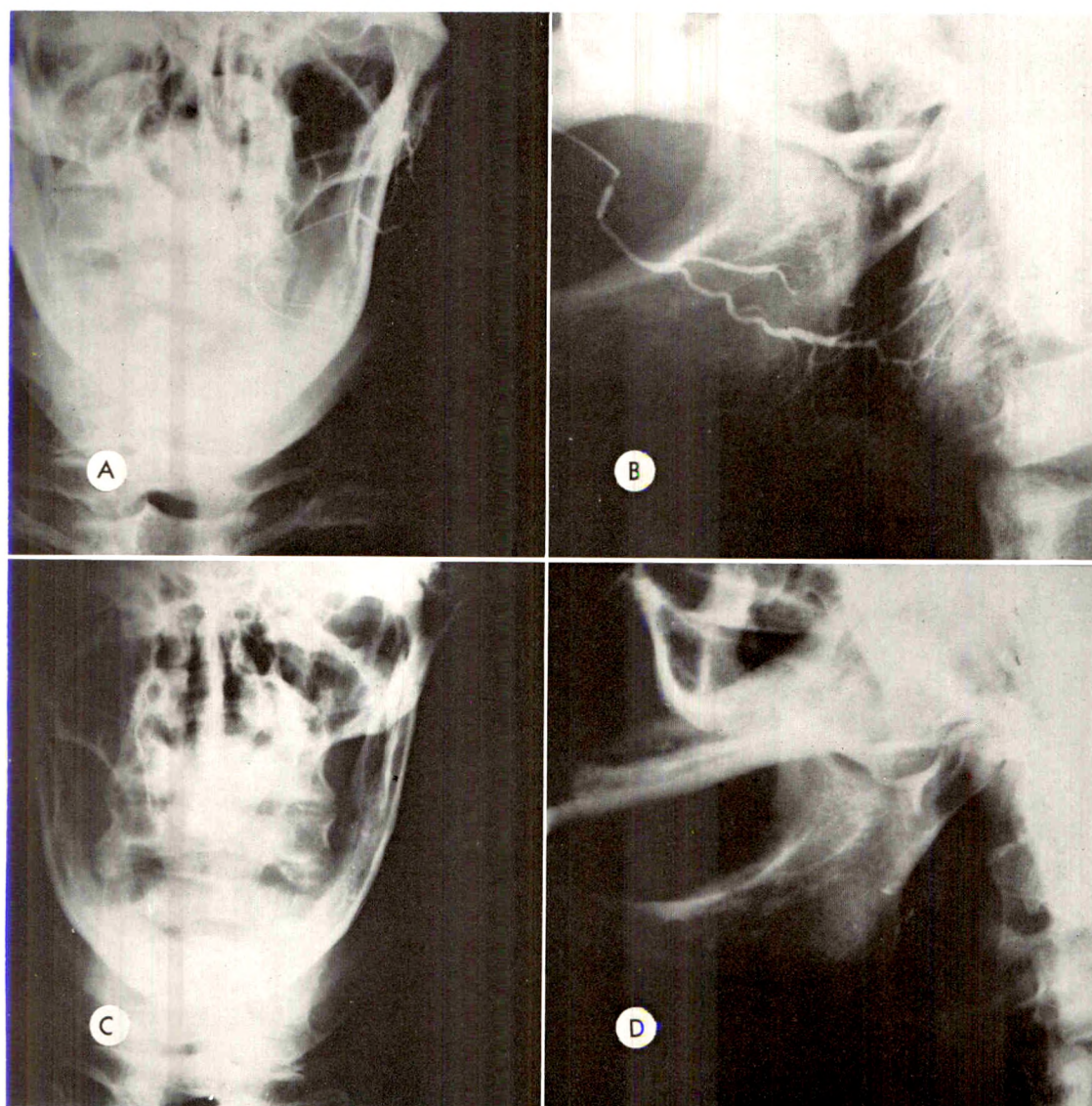


FIG. 9. Normal (pre-irradiation) sialograms in patient (J.H., 04-48-78). (A and B) The gland is of normal size and the filling extends into the minor ducts. (C and D) Evacuation is prompt and complete (5 minutes after stimulation).

without small duct and acinar filling (Fig. 11, A, B and C). In contrast to the immediate postirradiation sialograms, the gland size appeared to be within normal limits and evacuation was more nearly complete at 24 hours after introduction of the contrast medium.

Specific Identification of Serum Amylase. Using Weime's method of agar-gel electrophoresis²⁵ and the starch-iodine color reaction, we have been able to demonstrate

several distinct bands of amylolytic activity in serum and urine. The position of the amylolytic bands, in relation to the serum proteins, corresponded to those described by McGeachin and Lewis,¹⁶ who used paper electrophoresis and elution. Moreover, a difference in the electrophoretic mobility of the amylolytic components of pancreatic and salivary gland homogenates permits their differentiation.

A representative zymogram (R.P.,

04-81-95) is shown in Figure 12. The pre-irradiation serum sample was representative of those from a number of normal controls. Beginning with the third hour after irradiation, an intense amylolytic band appeared in the zone of the gamma globulins. The intensity of this band was proportional to the serum amylase activity.

Moreover, its electrophoretic position appeared to be identical with that of the amylolytic fraction obtained with homogenates of salivary glands. The appearance of this band was noted in all postirradiation sera studied.

Excretion of Amylase. Three patients, who received preoperative irradiation but

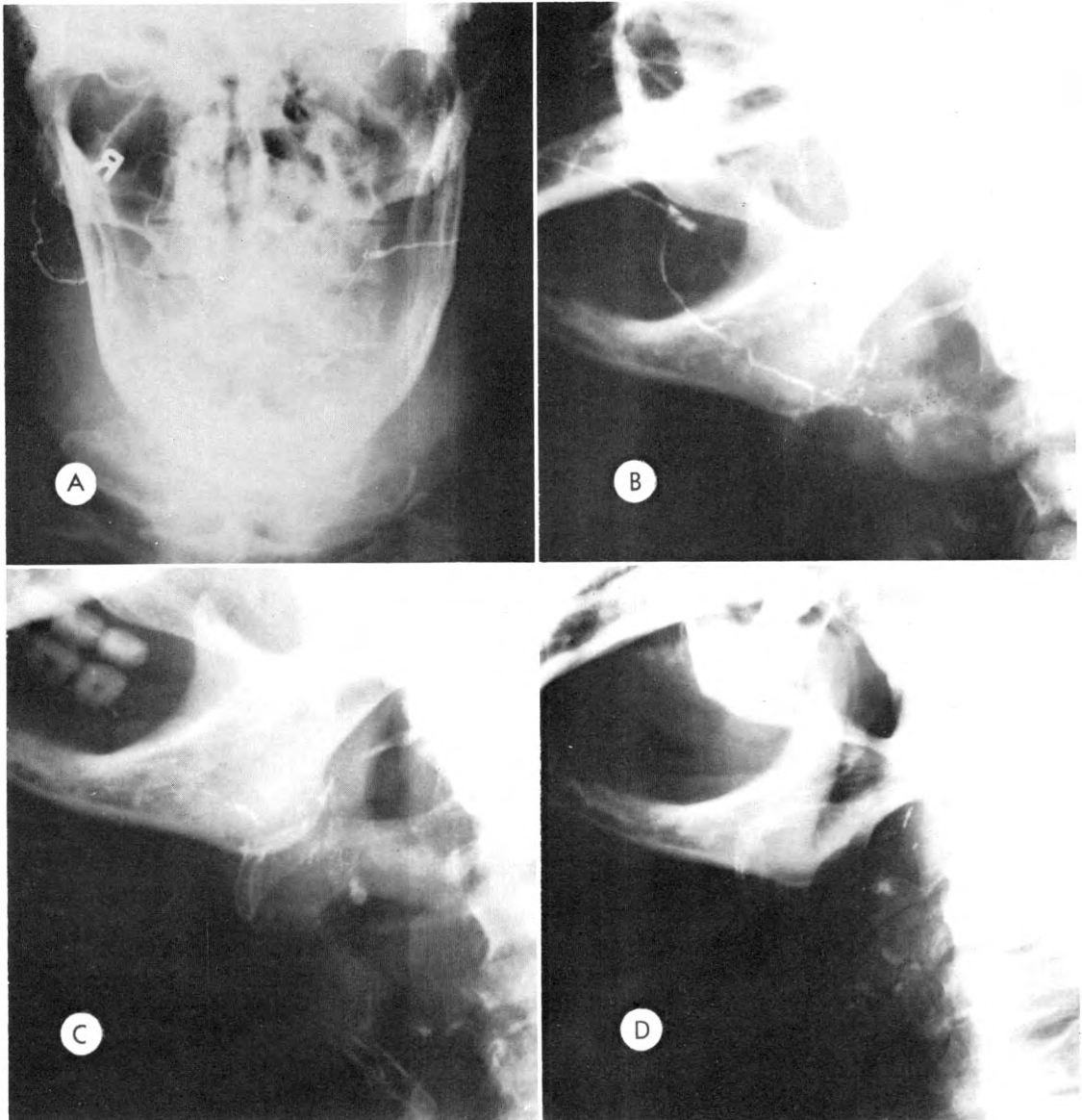


FIG. 10. Postirradiation sialograms. *A*, *B* and *C* taken 24 hours after 2,000 r. (*A* and *B*) There is pronounced soft tissue swelling due to parotid gland tumefaction, displacement of the principal ducts and incomplete filling. Contrast medium is not completely evacuated 5 minutes after stimulation (*C*) and radiopaque remnants are present 25 days later (*D*).

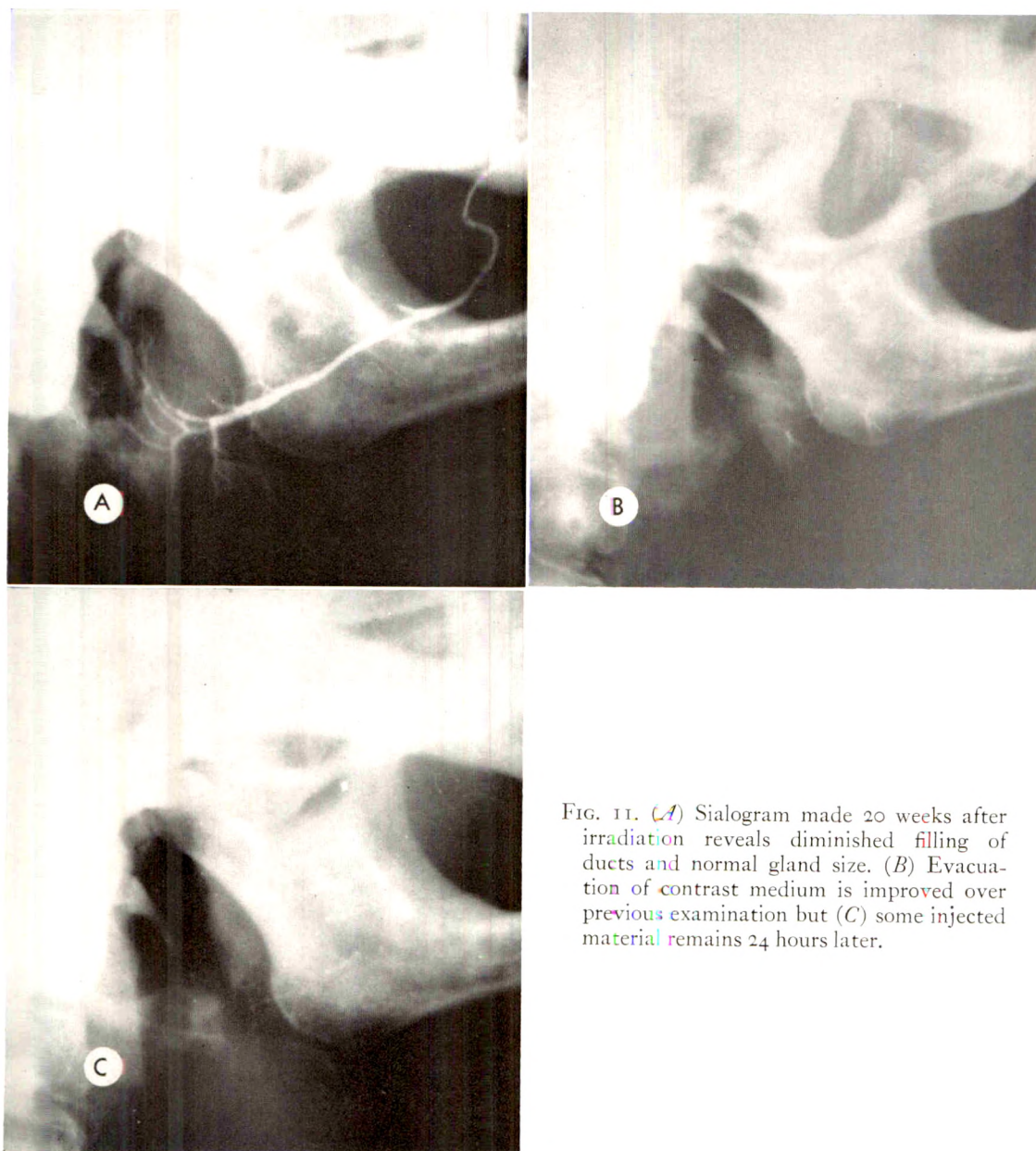


FIG. 11. (A) Sialogram made 20 weeks after irradiation reveals diminished filling of ducts and normal gland size. (B) Evacuation of contrast medium is improved over previous examination but (C) some injected material remains 24 hours later.

who are not listed in the previous groups, were studied to assess the urinary excretion of amylase. All had a marked rise in urinary amylase, both in terms of concentration and in total amylase excreted; this rise paralleled the rise of amylase in the serum following irradiation (Fig. 13). The major increase in urinary amylolytic activity was in the electrophoretic zone corresponding to

the "salivary amylase" observed in the serum.

DISCUSSION

CLINICAL RESPONSE

The clinical response to irradiation in these patients agrees closely with the descriptions previously set forth by Bergonie and Speder³ in 1911 and by Ceresole⁷ in

1912. The degree of tissue reaction is undoubtedly greater in the present group of patients, particularly in those receiving radiation in excess of 1,000 r. Ceresole noted these postirradiation sequelae in only 25 per cent of his patients, whereas in the present study salivary gland swelling was an invariable occurrence. This greater incidence of discernible reactivity of the salivary glands is undoubtedly related to the larger single tumor doses employed.

The early complaint of dryness of the mouth is probably attributable to a decrease in salivary flow after irradiation of the salivary glands. Although flow rates were not specifically measured, the failure to evacuate contrast medium during the immediate postirradiation sialographic study is suggestive of a sharply diminished salivation. Furthermore, persistence of radiopaque material within the duct system for at least 24 days points to some impairment of salivary flow. The histologic fea-

tures of fibrosis and acinar atrophy noted in salivary glands long after irradiation suggest that the xerostomia following irradiation is due to a decreased amount of functional salivary gland tissue.

In spite of histologic evidence of severe tissue damage and foci of suppuration in many of the glands, elevation of temperature was infrequent and a leukocytosis was rarely seen. There was no evidence in the histologic studies or the clinical course of the patients to suggest that a bacterial or viral agent had been activated. Indeed, unlike suppurative parotitis of bacterial origin, the reaction rapidly subsided without any specific therapy.

AMYLASE CHANGES

Numerous organs have been shown to contain amylases but the pancreas and the salivary glands are known to contain the largest amounts.¹⁵ Saliva is rich in this enzyme and it is stated that the parotid and

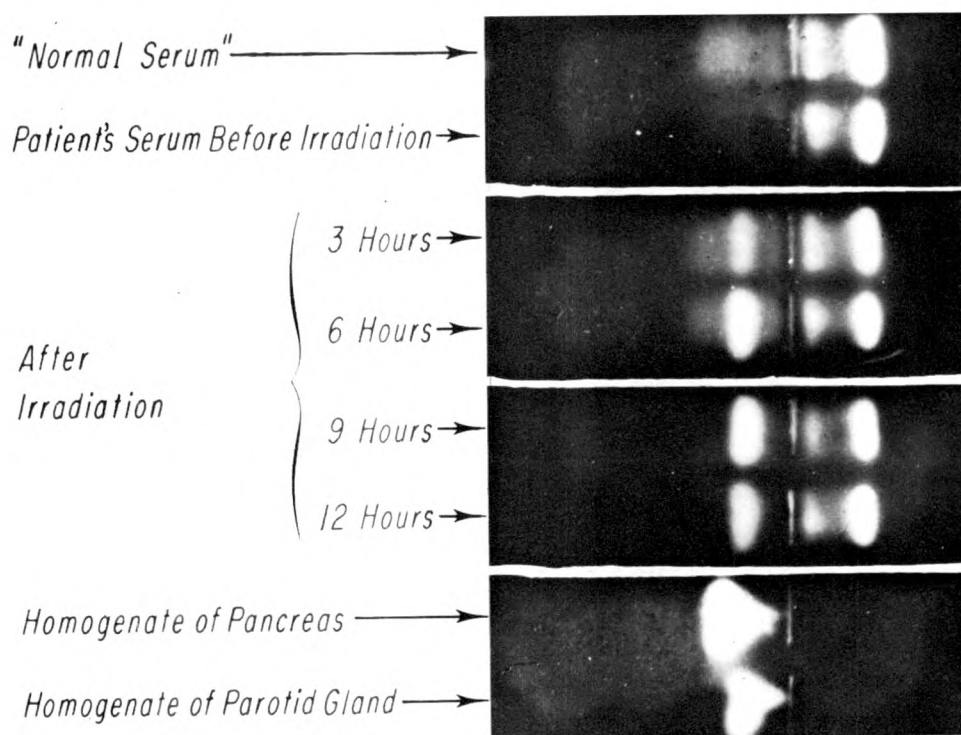


FIG. 12. Serial zymograms demonstrating the appearance of an intense amylolytic band in the postirradiation sera (R.P., 04-81-95). The zone which appears after irradiation corresponds to the amylolytic zone obtained from parotid gland homogenate. The latter is distinguishable from the pancreatic amylase.

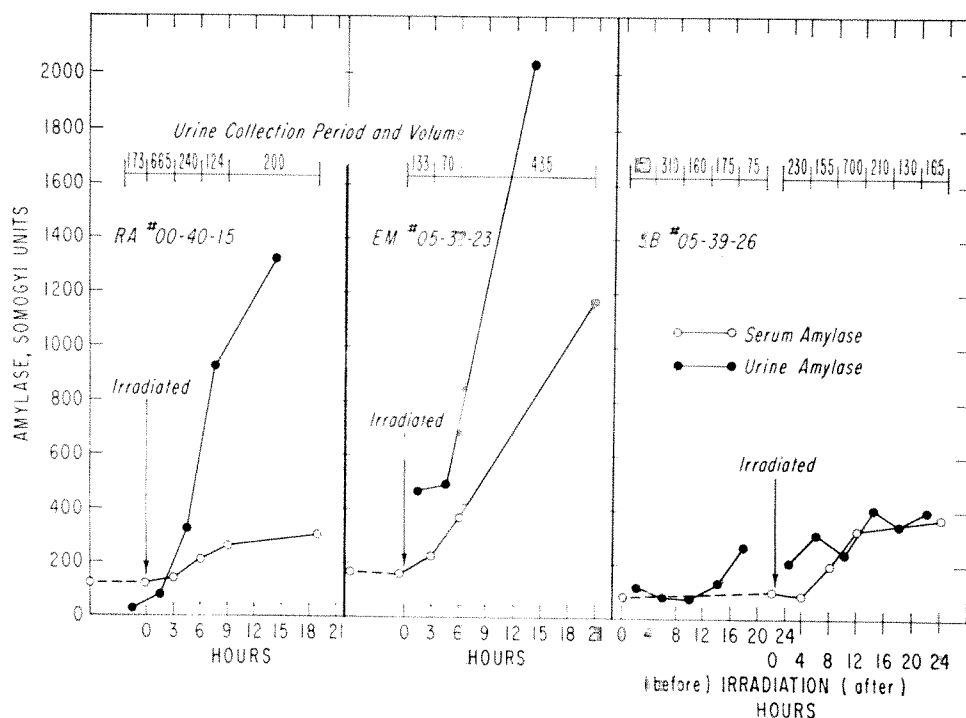


FIG. 13. Serum and urine amylase levels following irradiation of head and neck areas.

submaxillary saliva of the dog, cat, cow, horse, sheep and goat contains low levels of activity, whereas the saliva of man, ape, guinea pig, mouse, rat and rabbit has high levels of enzyme activity.⁵ Somogyi,²⁰ in a review of the literature in 1941, concluded that normally blood diastase does not originate either directly or indirectly from the salivary glands. He noted that inflammation of the salivary glands is accompanied by hyperamylasemia. This has been corroborated for epidemic parotitis^{6,24} and calculous obstruction of salivary gland ducts.¹² Clinically, hyperamylasemia has been particularly helpful in the diagnosis of inflammatory disease of the pancreas. Elevated serum amylase has also been reported in instances of perforated duodenal ulcer, intestinal obstruction, ruptured ectopic pregnancy and in some patients convalescing from laparotomy.¹⁴ Hyperamylasemia has been demonstrated following the administration of opiates.¹¹ The hospital records of the patients in the present study were reviewed and it was ascertained that such drugs were not used.

We feel that the following points support the conclusion that the irradiation of the salivary glands is causally related to hyperamylasemia.

1. Hyperamylasemia invariably follows irradiation of the salivary glands.
2. The degree of hyperamylasemia is directly related to the mass of salivary gland tissue irradiated.
3. The degree of hyperamylasemia is a dose-related phenomenon.
4. Serum enzyme changes were not noted in patients receiving regional irradiation to other parts of the body. We have made serial examinations of sera following chest and abdominal irradiation in several patients. We have particularly sought and failed to find any amylase rise following irradiation of the abdomen. It is generally agreed that the pancreas, an amylase-rich organ, is radioresistant.⁴
5. The serum amylase elevation is diminished when the salivary glands are repeatedly irradiated. The responses recorded in Figure 7 illustrates this relative refractoriness. Another patient, M.B. (03-17-20),

received 2,500 r to more than 50 per cent of the total salivary gland mass, yet the peak postirradiation serum amylase was only 481 Somogyi units, considerably less than the levels seen among all other patients who received the same amount of radiation. Four years earlier she had received 3,700 r for a malignant neck mass. Although we lack histologic proof of salivary gland atrophy in this case (no tissue obtained), it is significant that xerostomia was present at the time of her admission to the Clinical Center.

6. Hyperamylasemia resulted from irradiation of the parotid gland alone. The patient shown in Figure 8 received 100 r to less than 25 per cent of the total salivary gland mass. Despite this low dose, a rapid serum amylase elevation to 1,200 units resulted, illustrating the remarkable sensitivity of the serum amylase as an indicator of the action of radiation on the salivary glands.

7. In the postirradiation hyperamylasemia, there is an increase in a specific amylase component in serum identical with that from salivary glands. The electrophoretic studies constitute the most conclusive proof that the major increase in amylase in the serum is of salivary gland origin. It is clear, as illustrated in Figure 12, that the heightened amylase activity appears as a zone migrating with the globulins in the postirradiation serum samples, whereas no such zone was demonstrated in the preirradiation serum specimen. Moreover, the intensity of the amylase activity of this zone parallels the increase in total activity of the serum and the zone corresponds in mobility to the amylase zone obtained with a homogenate of salivary gland tissue.

The hyperamylasemia in the irradiated patients was transient as is that in acute pancreatitis and epidemic parotitis.¹⁷ In the three patients studied and summarized in Figure 13, it is evident that the amylase level of urine parallels and exceeds that of serum in the postirradiated patient. Furthermore, the increase in urinary amylase

is, for the most part, present in the same electrophoretic zone previously noted to represent salivary gland amylase. Thus urine amylase levels rapidly reflect the hyperamylasemia of postirradiation sialadenitis.

HISTOPATHOLOGY

The histopathologic changes observed in the acutely irradiated salivary glands are in harmony with the clinical features of glandular swelling and hyperamylasemia. A review of salivary glands present in an additional 82 radical neck dissection specimens has failed to reveal acute changes in any way comparable to those observed in these preoperatively irradiated patients. The single case in which the submaxillary gland showed no histologic reaction is noteworthy because the parotid gland from the same surgical specimen displayed a typical acute reaction. The irradiation port in this case was such that the parotid gland was in the field, whereas the submaxillary gland was not. The findings in this case emphasize that the acute change is evoked only by direct irradiation. The mechanism of the tissue reaction is unexplained but the observation that the serous cells exhibit the greatest damage suggests that it is in these cells that the primary damage occurs. Actual disruption of cells or changes in cell permeability with release of intracellular amylase into the circulation would seem to account for the appearance of the salivary amylase in the serum. The marked inflammatory cell infiltration into the glands appears to be a response to serous cell degeneration.

The inflammation and degeneration in the irradiated salivary glands represent an early or acute reaction. The histopathologic features of the glands suggest in all cases that a common tissue response occurred and varied only in degree. From the pattern of reaction seen at 24 hours after irradiation, we visualize a progressive change in which there are continued degeneration and loss of acini, replacement of acute inflam-

matory cells by chronic inflammatory cells, fibrosis within lobules, and persistence of duct structure resulting in the chronically altered gland frequently seen by pathologists but seldom described in detail. The salivary glands of 2 patients reported by Van Salis²³ and the few cases reported by Evans and Ackerman¹⁰ were all obtained 10 or more days after irradiation. In general these glands were made up of a duct system with few, if any, acini remaining. There were interlobular and periductal fibrosis and some chronic inflammatory cells in the lobules. A few previously irradiated salivary glands in our files display these same features.

An experimental animal as a model for studying the reaction of salivary glands to irradiation is desirable but reports of studies on animals (mainly histologic studies) indicate that the salivary glands of animals are less apt to give the acute change than are the glands of humans.^{9,18} Few detailed studies of salivary glands have been made within the first 24 hours after irradiation; perhaps a review of the responsiveness of animal salivary glands will reveal greater reactivity than seems to be indicated in published reports.

The principal conclusions from the limited observations in sialography are those that previously have been described, namely, that the prompt and near total cessation of salivary gland secretion occurs early in the postirradiation period. There appears to be subsequent (partial?) recovery of this function. These views are in accord with the observation that the rate of evacuation of contrast material from the gland, although not quantitative, is a relative index of salivary flow. The more rapid evacuation during the study done 20 weeks after irradiation may represent a limited recovery of glandular function. The chronic and apparently permanent damage sustained by these glands is manifest by the incomplete filling of the gland, the smaller gland volume and the absence of acinar patterns; these findings are consistent with

the histologic features of atrophy in late stage glands.

AMYLASE AS A BIOLOGIC INDICATOR OF RADIATION EXPOSURE

The present report reveals that serum amylase levels are related to the dose of radiation and the amount of salivary gland tissue irradiated. The radiation in each instance was localized to head and neck fields but at least two reports from the literature indicate that salivary glands react similarly in whole body irradiation. Two of the 9 men accidentally exposed to ionizing radiation from a klystron tube at Lockport, New York, in March, 1960 are reported to have had temporo-mandibular tenderness as an early symptom. Of special interest is the case of K.S., who was seen by his own physician on the morning of the radiation exposure and, on the basis of the parotid swelling, was diagnosed as having mumps.¹³ Unfortunately, serum amylase determinations were not included in the report. The estimated dose is thought to have been as "high as 1,200 to 1,500 rads over certain areas of the body." Both men received radiation to the head and trunk.

A second subject is reported by Thomas *et al.*²¹ Their Case 5, a 38 year old man with acute leukemia, received whole-body irradiation prior to a bone marrow infusion. The air exposure from 2 cobalt 60 sources was 2,016 r and the calculated tissue exposure 1,596 r; the exposure rate was 2.4 r per minute. Serial amylase determinations in this patient revealed a sharp increase, reaching a high point of 1,600 Somogyi units at 24 hours, and returning promptly to normal levels over the succeeding several days. Unfortunately, specific mention of parotid swelling or abdominal pain is not included in the report. To our knowledge this is the only prior documented instance of a recorded radiation-induced hyperamylasemia. The general pattern of response in this single case conforms closely with our observations.

The foregoing cases from the literature

suggest that serum (salivary?) amylase elevation may be expected in human subjects exposed to whole body irradiation. Serum chemical changes, other than those of amylase, have been inconstant and appear to depend on the vagaries of the clinical courses of the respective radiation-accident victims. Although a general statement regarding the utility of amylase as a biologic indicator of radiation exposure is premature, further observations would seem worthwhile. The determinations of serum and urinary amylase are readily available in most clinical laboratories.

CONCLUSIONS

The clinical, serum chemical and microscopic features of an acute postirradiation sialadenitis have been described. To us, the most significant finding appears to be the elevation of serum and urinary amylase, or more specifically, elevation of the salivary amylase component of serum and urine. To our knowledge, this numbers among the few biochemical changes that occur early and constantly following irradiation. Our experience indicates that direct exposure of the salivary glands is necessary to provoke this change, and that the serum amylase response is related to the dose of irradiation.

The experience from this study and at least 2 instances from the literature suggest that the appearance of the salivary amylase in serum may be the basis for development of a test useful in screening human subjects accidentally or deliberately exposed to radiation. The further thought that these observations can be utilized to investigate more minute details of radiation damage and to study recovery from radiation damage is most appealing.

SUMMARY

1. The acute clinical picture which results from irradiation of the salivary glands consists of transient swelling of these glands, generally unaccompanied by fever or leukocytosis.

2. Evidence is presented which impli-

cates the salivary glands as the tissues of origin for the postirradiation hyperamylasemia.

3. The acute histopathology of the irradiated human salivary glands is presented and the clinicopathologic correlation is discussed.

4. The possible usefulness of hyperamylasemia as a biologic indicator of radiation exposure is suggested.

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ADDENDUM

Since submission of this paper a careful and detailed study of lesions of salivary glands and teeth following irradiation appeared (R. M. FRANK, J. HERDLY, and E. PHILIPPE, *J. Amer. Dent. Ass.*, 1965, 70, 868). The authors relate irradiation of the salivary glands to progressive involutional changes in the glands and reduced salivary flow—findings entirely consistent with our observations. In addition, they observed that saliva was qualitatively altered following irradiation of the salivary glands and that certain dental lesions occurring after irradiation of fields which included the salivary glands did not occur when only the teeth were irradiated.

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JUVENILE NASOPHARYNGEAL FIBROMA*

ROENTGENOLOGIC CHARACTERISTICS

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DURING a recent study of the roentgenologic manifestations of malignant tumors of the nasopharynx by Miller,¹⁸ some cases of juvenile nasopharyngeal fibroma were reviewed in the course of the preparation of a differential diagnosis. It was noted that an unusual roentgenologic appearance was demonstrated in a sufficiently large number of such cases to warrant a separate study.

Recognition of a nasopharyngeal fibroma is important because preliminary biopsy of such lesions may be fraught with considerable danger owing to profuse hemorrhage which is often difficult to control.^{4,18} If the preoperative diagnosis can be made with reasonable certainty, such a danger may be avoided.

There are many reports in the literature concerning this type of lesion, but apparently there has been some uncertainty as to whether all of the cases reported actually represent the same entity. Thus, for example, not all of the tumors reported by Handousa, Farid and Elwi¹⁰ in a clinicopathologic study of 70 cases would fit the definition of this condition as implied by certain other authors.^{15,19} The data herewith reported are associated with lesions which seem to satisfy the following definition given by Martin and co-workers¹⁵ in 1948: "Juvenile nasopharyngeal angiofibroma is a specific, highly vascular, noninfiltrating, essentially benign neoplasm, occurring in the nasopharynx or posterior nasal cavity of pubescent males."

The roentgenograms of 46 patients who had such lesions verified by competent pathologists were studied and a fairly typical roentgenographic pattern emerged

which seemed to aid in the differentiation of these lesions from other tumors in this anatomic region.

A reasonably complete survey of the literature failed to reveal any pathognomonic roentgenologic sign of this interesting condition despite the fact that most of the features have been indicated in various reports.^{12,17}

Although generally regarded as a rare tumor,⁴ it must have been recognized for a long time because Tapia Acuña²¹ quoted Celsus in *De Arte Medica* as having credited Hippocrates with removing such a lesion. Most descriptions of these tumors have been in papers concerned primarily with treatment^{3,4,6,9,11,14,16,20} and these reports are rather uniform in stating that generally such lesions are discovered in adolescent boys⁶ although there are isolated case reports of these lesions in young women.^{1,7} These tumors usually regress after about the age of 20 to 25 years or when sexual maturity is reached. Complete regression may not be the rule. They are benign in the sense that most authors believe that metastasis does not occur. However, they can be very dangerous, expanding as they do in the region of the base of the skull, near important blood vessels and nerves and in a manner which frequently obstructs the airway.^{4,6,11} They do not invade adjacent structures by cellular infiltration but erode by pressure due to their relentless expanding type of growth.^{2,4,5,6,21} In a unique case reported by Massoud and Awwad,¹⁷ metastasis to cervical lymph nodes and possibly to the lumbar spine is said to have occurred.

These tumors are usually attached to the

* Presented at the Sixty-fifth Annual Meeting of the American Roentgen Ray Society, Minneapolis, Minnesota, September 29 to October 2, 1964.

roof of the nasopharynx, frequently near the nasal septum, and they grow slowly into the pharynx and the nasal fossa.¹⁵ Patients usually have some degree of nasal obstruction but ordinarily complain little until hemorrhage occurs. Occasionally, other symptoms are noted when structures such as the paranasal sinuses or the orbit are eroded.^{8,14}

Recognition is important because a single biopsy procedure may result in uncontrolled bleeding and thereby precipitate a grave crisis. On the other hand, since these tumors are rather rare and inasmuch as there are other, more benign, masses in this region such as ordinary fibroma and nasal polyp, it would seem helpful to be able to make a reasonably definite roentgenologic diagnosis. This apparently can be done by carotid angiography when necessary,²² and this is preferable to uncontrolled bleeding due to biopsy. However, the observations about to be described seem to offer reliable aid in diagnosis as well as in assessing the size and extent of the tumor.

ROENTGENOGRAPHIC FEATURES

In this study of the roentgenograms of 46 patients in whom a juvenile nasopharyngeal fibroma was found and proved, the patients were from 8 to 24 years old (mean 14 years). All were males and all complained of one or more of the following symptoms: episodes of severe bleeding from the nose, nasal obstruction, and a mass in the nose and pharynx. Nineteen complained primarily of severe epistaxis. Thirty patients complained of an obstruction in at least one side of the nose and 13 complained of a mass in the nose or pharynx. Sixteen of the 46 patients complained of more than one symptom.

The following roentgenograms were available for study: roentgenograms of the paranasal sinuses in 42 cases, roentgenograms of the head in 8, submentovertical stereoscopic views in 19, laminagrams of the sphenoid region in 8, and roentgenograms of the facial bones made by the Bucky technique in 17.

The roentgenograms of 45 of these 46 patients showed a large soft-tissue mass in the nasopharynx which extended into areas of parapharyngeal structures. A significant part of each mass was well delineated by an adjacent air shadow in every case (Fig. 1, *a*, *b* and *c*). These sharply circumscribed portions occurred within the pharynx in 45 cases, but were also apparent in the sphenoid sinuses in 28 (Fig. 1*a*), the nose in 13, the maxillary antrum in 20 (Fig. 1*b*), the ethmoid sinus in 16 (Fig. 1*c*), and the frontal sinus in 1. The mass extended well into the soft tissue of the cheek in 2 instances and also into the orbit in 2.

A characteristic feature occurred in 40 of 46 cases. This consisted of an anterior bowing of the posterior wall of the maxillary antrum of the involved side which was best seen in the lateral projection but, occasionally, also appeared in the submentovertical view. These patients, therefore, had a reversal of the usual convex posterior contour of the posterior wall of the antrum. The extent of this forward bowing was estimated in each case and 3 groups were thereby determined. Group 1 (Fig. 2*a*) contained 3 cases and included those instances in which the forward bowing appeared to be less than 5 mm. in depth. The curve was judged by comparing it with that of the opposite side. Group 2 (Fig. 2*b*) contained 18 cases and included those patients in whom the forward bowing measured from 5 to 10 mm. as compared with the normal side. In 1 of these, the bone margin was so thin that it was difficult to distinguish it from the outline of the soft-tissue mass behind. Group 3 (Fig. 2, *c* and *d*) contained 19 cases and included those in which the forward bowing of the antral wall was 10 mm. or more.

Because this antral sign appeared unique, the roentgenograms in 240 proved cases of other types of nasopharyngeal and oropharyngeal tumors were reviewed. In only 1 of these 240 cases was there seen a posterior antral wall contour which in any way resembled that seen in so many of the juvenile nasopharyngeal fibromas. This is

not to say that other types of tumors could not possibly produce such a roentgenologic sign but it must be exceedingly rare except as associated with the juvenile nasopharyngeal fibromas under discussion. For example, an illustration in an article by Jönsson¹² shows what could be interpreted as a Group 1 antral sign (page 654, Fig. 3) associated with an undifferentiated tumor. The legend states that it "resembles a pharyngeal fibroma, but as the patient is an older woman, this diagnosis is excluded."

The roentgenologic evidence of erosion of bone adjacent to these nasopharyngeal tumors in 30 cases of this series was studied. The bone changes differ from the invasive destruction of most neoplasms in that the bone margins retain a rather sharp and usu-

ally a rather dense appearance even though a considerable amount of the bone has been destroyed. This type of erosion is that seen in association with expanding aneurysms, neurofibromas and other tumefactive processes which expand slowly and are fairly smooth in outline.

In 10 of the 46 cases a previous operation, either biopsy or partial removal, had been done and in each of these cases radon seeds had been implanted into the tumor. These procedures may have affected the appearance of the erosion in 5 instances but probably did not have any effect on the appearance of bone structures in the other 5. In 7 instances no definite bone erosion was obvious on examination of the roentgenograms. In only 1 of these was a submento-

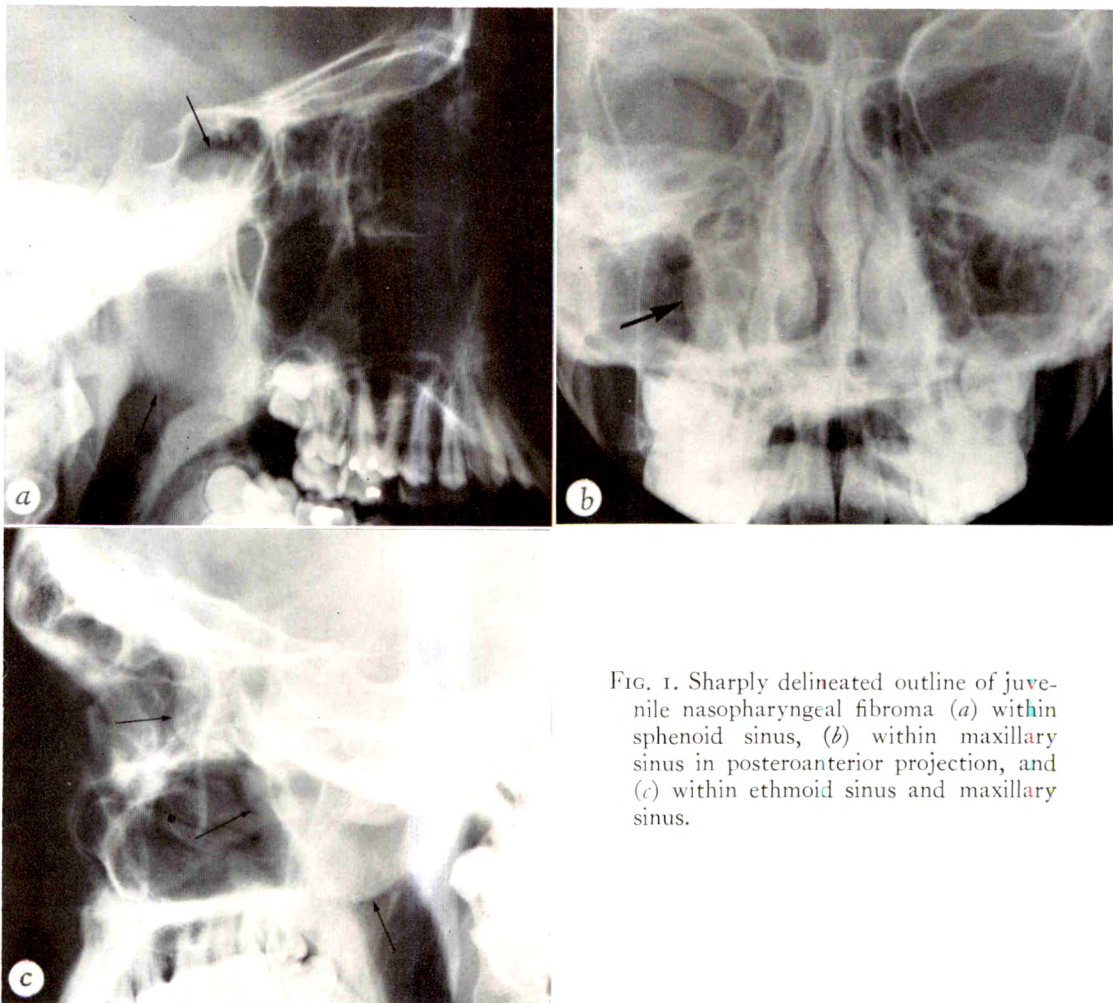


FIG. 1. Sharply delineated outline of juvenile nasopharyngeal fibroma (a) within sphenoid sinus, (b) within maxillary sinus in posteroanterior projection, and (c) within ethmoid sinus and maxillary sinus.

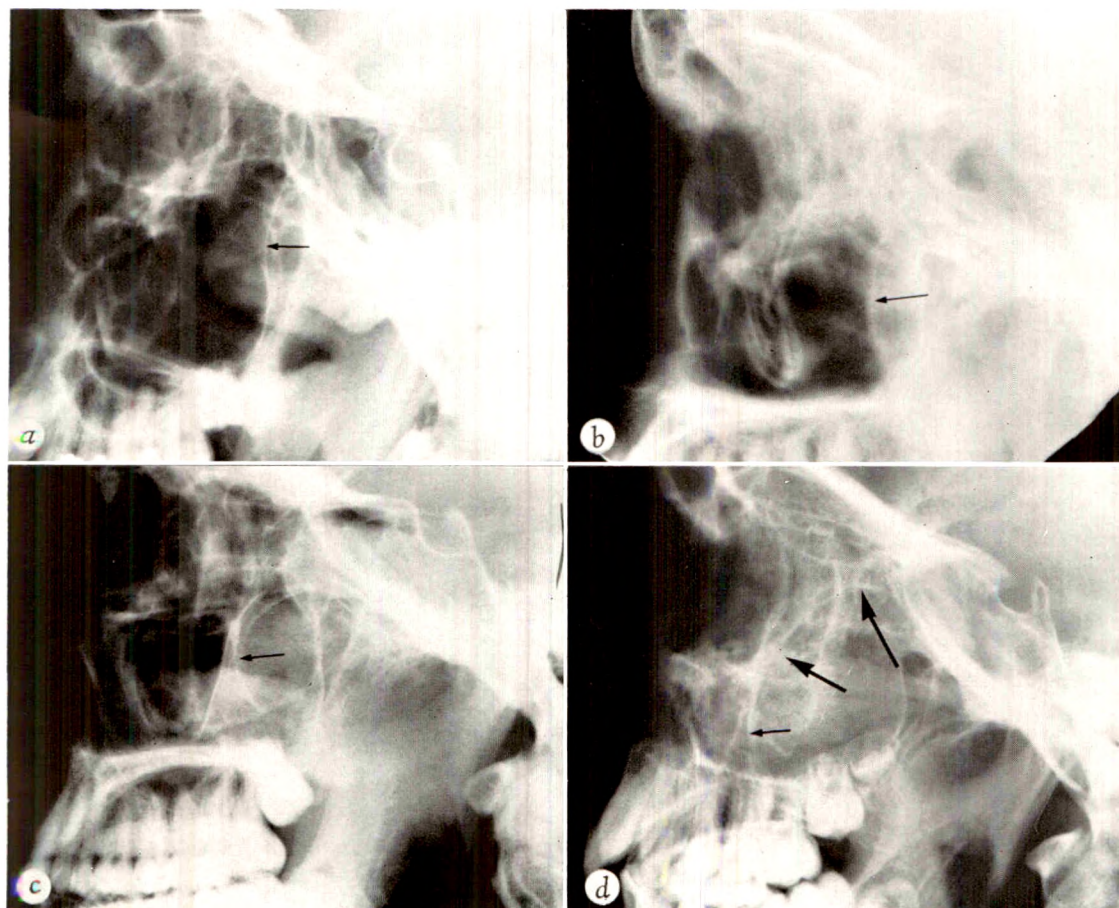


FIG. 2. "Antral sign." Anterior bowing of posterior maxillary antral wall: (a) Group 1, (b) Group 2, and (c and d) Group 3.

vertical view available, and in the others only roentgenograms of the paranasal sinuses were available for interpretation. In 2 of the cases the roentgenograms were of insufficient quality to show bone structures and in 1 (the case in which no well-demarcated mass was found) the diffuse density of the nasopharynx and paranasal sinuses precluded any judgment concerning bone erosion. In 1 other case there had been a more extensive operation and therefore erosion was not possible to evaluate.

The most common erosive change demonstrated involved the sphenoid bone. In 30 cases definite bone erosion of the inferior portion of the body of the sphenoid was apparent. In 19 of these there was what might be termed a "sphenoid hole" (Fig. 3). This is a rather sharply circumscribed, fairly

round radiolucent defect in the inferior portion of the body of the sphenoid and this defect is surrounded by a well-circumscribed and fairly dense bone margin. It is apparently through this hole that the nasopharyngeal fibroma enters the sphenoid sinuses. In 11 cases the sphenoid sinus was eroded from below, but a definite hole into this sinus could not be recognized (Fig. 4). Erosion always occurred above the sphenoid-occipital synchondrosis.

In 18 instances there was obvious bone erosion of the hard palate on one or both sides. This usually involved a portion of the vomer and extended into the nasal fossa.

In 13 cases there was definite erosion of the region of one or both of the pterygoid fossae and also erosion of a portion of the hard palate (Fig. 5).

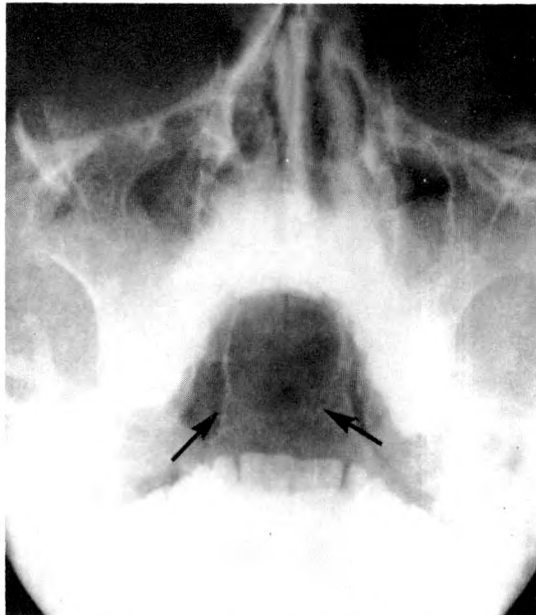


FIG. 3. Sharply circumscribed defect in body of sphenoid through which tumor extended into sphenoid sinus ("sphenoid hole").

In 2 instances it was possible to visualize erosion of the medial wall of the maxillary sinus due to the portion of the tumor in the nasal fossa. In 1 instance the floor of the sella turcica was eroded and the tumor obviously extended up into this fossa. In 2 cases the density of the tumor was so diffuse that it was difficult to see the sharp margins of the bone erosion, although it was evident that part of the floor of the middle fossa was eroded.

In 21 of the 46 cases, there was some deviation of the nasal septum associated with the tumor. This was seen on the Caldwell (Fig. 6) and submentovertical projections in most instances although, occasionally, it was also demonstrated in the Walters' views.

An attempt was made to determine possible changes involving the wings of the sphenoid bone in the region of the superior orbital fissure. In 43 instances the superior orbital fissures were demonstrated sufficiently well for evaluation. Of these, there was definite enlargement of one of the superior orbital fissures in 16 instances (Fig. 6). This enlargement usually took place in

the lower portion of the fissure and involved its outer margin, which, although eroded laterally, maintained a rather hard, dense margin. It was possible to see the foramen rotundum in 18 of the 43 cases in which the superior orbital fissure was demonstrated. In 2 of these, there were enlargement and relative decalcification of the wall of the foramen rotundum immediately below the involved superior orbital fissure.

COMMENT

This study of 46 proved cases of juvenile nasopharyngeal fibroma seems to indicate that there are roentgenologic features which, when taken together with the clinical evaluation of the patient, make possible a reliable tentative diagnosis in a large number of cases. It indicates that it would be prudent and advisable to make sinus and submentovertical roentgenograms on all male patients between the ages of 8 and 25 who have one or more of the following manifestations: episodes of severe bleeding from the nose, nasal obstruction as a

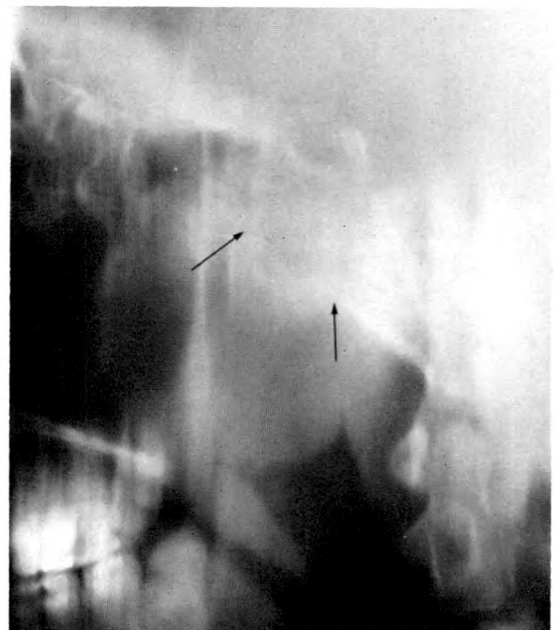


FIG. 4. Lateral laminagram showing sharply circumscribed juvenile nasopharyngeal fibroma extending within sphenoid sinus.

chronic complaint, or a tumefactive mass in the nasal fossa or nasopharynx. This should be done before any attempt at biopsy or treatment is undertaken. There is a very great probability that should a nasopharyngeal fibroma be present it will have produced some erosion of the bone structures immediately adjacent to a well-demarcated soft-tissue mass and probably would show the so-called antral sign of anterior bulging of the posterior wall of the maxillary sinus in the lateral view. Maxillofacial surgeons who operate on these tumors know that these masses frequently grow behind the antrum, although the roentgenologic features of this type of growth have not been emphasized. Since this roentgenologic feature appears to be so very rare in other types of nasopharyngeal or oropharyngeal tumefactive processes and so characteristic in these cases, it would seem that one could proceed with a provisional diagnosis of nasopharyngeal fibroma without biopsy and only undertake

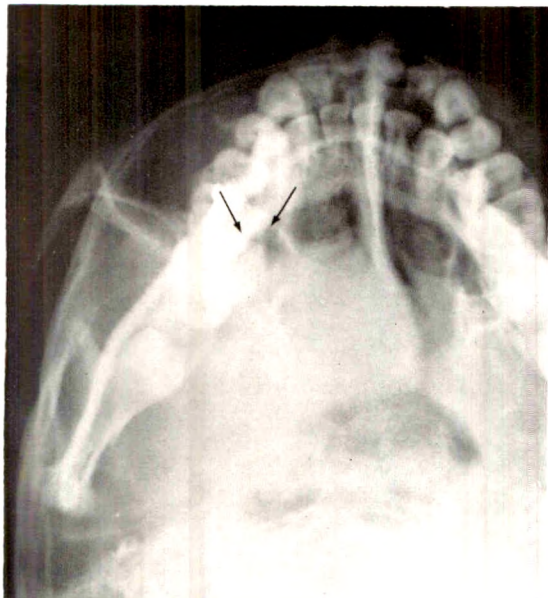


FIG. 5. Submentovertical projection showing large nasopharyngeal mass with erosion of portions of hard palate, pterygoid fossa and antrum on right side and with tumor extending from left of midline into right infratemporal fossa. Anterior bulging of posterior wall of right antrum is demonstrated.

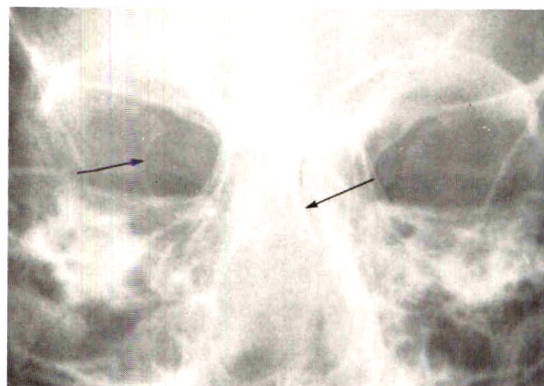


FIG. 6. Enlargement of superior orbital fissure due to erosion of lateral and inferior aspects. A sharp dense margin is maintained. Nasal septum is deviated by tumor in nasal fossa.

removal of the tumor after proper preparation of the patient, and in a place where the operation may be carried on with due regard for possible extensive hemorrhage. In the unusual instance in which these features are not present, it would seem prudent to undertake external carotid angiography as recommended by Thibaut²² to demonstrate the tumor before any attempt is made at biopsy.

No attempt has been made in this presentation to evaluate the choice of treatment once the diagnosis is definitely established. However, it can be reported that of these 46 patients with proved tumor of this type, 10 had had previous treatment with some form of radiation and the tumors had recurred. In several other cases the tumors recurred after inadequate removal. The literature indicates that these tumors usually regress to some degree in time, but the danger of hemorrhage, the difficulties associated with obstruction and the very great number of them that are associated with extensive bone erosions of the base of the skull indicate that whenever possible the tumor should be removed.

SUMMARY

The roentgenologic features, not including special contrast studies, of 46 cases of juvenile nasopharyngeal fibroma are described. A definite anterior bowing of the

posterior wall of the maxillary sinus was discovered to be present in 40 of 46 cases. This feature was not definitely identified in another review of 240 nasopharyngeal tumefactive lesions of other types and so, if present, would seem to be highly suggestive of the presence of nasopharyngeal juvenile fibroma. A somewhat characteristic bone erosion of the adjacent structures near the nasopharyngeal tumor was found in 30 of the 46 cases. This erosion almost invariably showed bone destruction, the limits of which were rather sharply demarcated by a well-delineated bone margin which was usually somewhat sclerotic. In 16 of the cases, the superior orbital fissure on the side of the tumor was enlarged in its inferior and lateral portion. In all cases, there was a large soft-tissue mass in the nasopharynx and oropharynx and in all but 1 instance the soft-tissue mass was well circumscribed where it appeared in one of the sinuses or in the nasal fossa. Since all of these patients were males between the ages of 8 and 24, it would seem that in such persons presenting a history of severe nasal bleeding, nasal obstruction, or a soft-tissue mass in the nasopharynx, when accompanied by these roentgenologic features, a rather confident diagnosis of nasopharyngeal juvenile fibroma can be made.

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THE EVALUATION OF PHARYNGEAL NEUROMUSCULAR DISORDERS BY CINEFLUOROGRAPHY*

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DYSPHAGIA localized to the hypopharynx and upper esophagus presents problems among the most difficult in diagnostic radiology. Conventional roentgenographic techniques are often inadequate to characterize the disorder. This paper describes the use of cinefluorography in the evaluation of neuromuscular disease involving the pharynx and upper esophagus.

While normal deglutition has been extensively studied in recent years,^{1,2} neuromuscular disorders of the pharynx have received little attention. Dahm⁴ in 1941 reported his studies of normal and disturbed pharyngeal deglutition, examined by means of kymography. This roentgenographic technique was employed to record motion during a limited time interval. The restrictions inherent in this technique diminished its value, although basic information was obtained contributing to the differential diagnosis between obstructive and paralytic pharyngeal dilatation. With the advent of cinefluorography, normal swallowing, the function and appearance of the cricopharyngeal muscle and the swallowing defect after bulbar poliomyelitis and in myasthenia gravis have been investigated.⁵ In addition, cinefluorography has been helpful in distinguishing between organic and neuromuscular disorders. However, pharyngeal weakness and paralysis are associated with a variety of vastly different neurologic lesions,³ and attempts to establish roentgenologic manifestations characteristic of one disease or of related groups of diseases have not been fruitful by this technique alone.

The recently successful application of simultaneous cinefluorography and manometry in the study of normal and disturbed esophageal motility suggested its use in patients with pharyngeal motor disturbance. This technique was employed in the majority of our patients under investigation for pharyngeal dysphagia and contributed greatly to the interpretation of the roentgenographic observations and the characterization of the various disorders in neuromuscular disease.

NORMAL DEGLUTITION

It may be useful to describe the normal sequence of motor activity in deglutition in some detail (Fig. 1). In neuromuscular disease, deviation from either the order or character of physiologic movement during the oropharyngeal stage of swallowing may occur. These defects will invariably lead to abnormal deglutition and may give rise to dysphagia. Employing cinefluorography and manometric techniques, the deviations from normal deglutition may be identified and analyzed.

1. The act of swallowing begins with movements of the tongue, to transport the food bolus towards the pharynx. As soon as the swallowing reflex is initiated, the mylohyoides, geniohyoides and digastricus muscles in the floor of the mouth elevate the tongue toward the hard palate.

2. The root of the tongue is then pressed against the posterior pharyngeal wall by the stylo- and hyoglossus muscles.

3. Thereupon, elevation of the soft palate by the levator and tensor veli palatini muscles takes place with the structure

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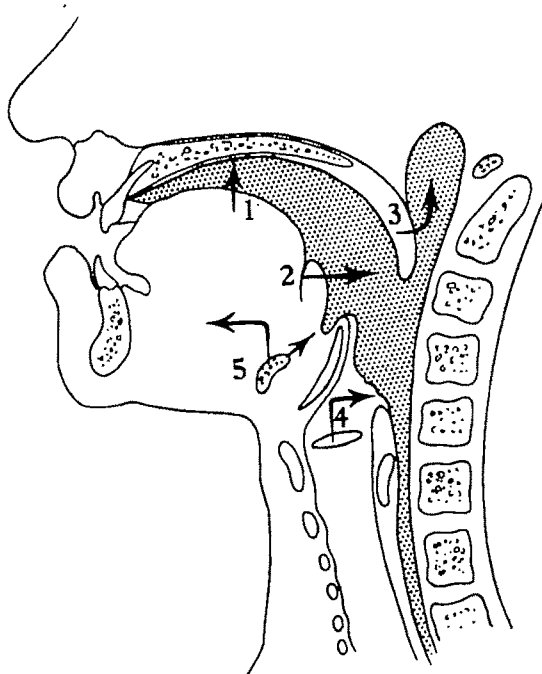


FIG. 1. Diagram of normal motor activity in the oropharynx (see text).

extending well above the level of the hard palate.

4. The larynx is then elevated and the hypopharynx is narrowed by the action of middle and inferior pharyngeal constrictors. In the propulsion of the bolus into the esophagus, it is noteworthy that contraction of the pharyngeal muscles occurs but peristalsis does not take place. Participation of the posterior wall of the pharynx during the act of swallowing is minimal.

5. Finally, tilting of the epiglottis occurs, brought about by contractions of the musculature of the floor of the mouth, and the hyoid bone is elevated upwards and posteriorly in a rapid anterior and backward motion.

Because of the rapidity of normal pharyngeal deglutition, pharyngeal structures are demonstrated only transiently during fluoroscopy. When recorded on motion picture film, however, they may be studied by frame-by-frame analysis to reveal even minor morphologic and physiologic changes from the normal.

During the cinefluorographic observation

of the swallowing process, the following anatomic landmarks of the pharynx may be observed: the pharyngeal recesses, *i.e.*, the valleculae and piriform sinuses, the aryepiglottic folds, the epiglottis, the vocal cords and laryngeal vestibule and the cricopharyngeal sphincter (Fig. 2; and 3, A-P).

THE CINEFLUOROGRAPHIC SIGNS OF PATHOLOGIC DEGLUTITION IN THE OROPHARYNGEAL STAGE

These can be divided into the following categories: 1. disturbance of motility; 2. residual filling of recesses, "vallecular-sign"; 3. stasis; and 4. misdirected swallowing (aspiration).

1. *Motility disturbance* is the most frequent sign of abnormal deglutition and the most reliable sign of pharyngeal paralysis. One can distinguish changes, both in nature and degree. These include impairment or complete loss of certain movements and/or a disordered sequence of functional activity. The latter may be due to a delayed initiation or prolonged duration of muscular activity, or a disturbance in the sequential order of the swallowing process. Motility is restricted if repeated attempts at swallowing are made before the act is actually carried out. The base of the tongue may move backwards several times until the swallowing reflex is initiated. Prolonga-

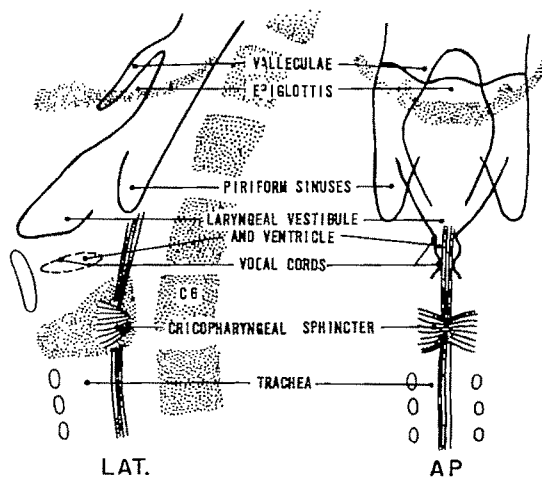


FIG. 2. Roentgenographic landmarks of the pharynx and larynx.

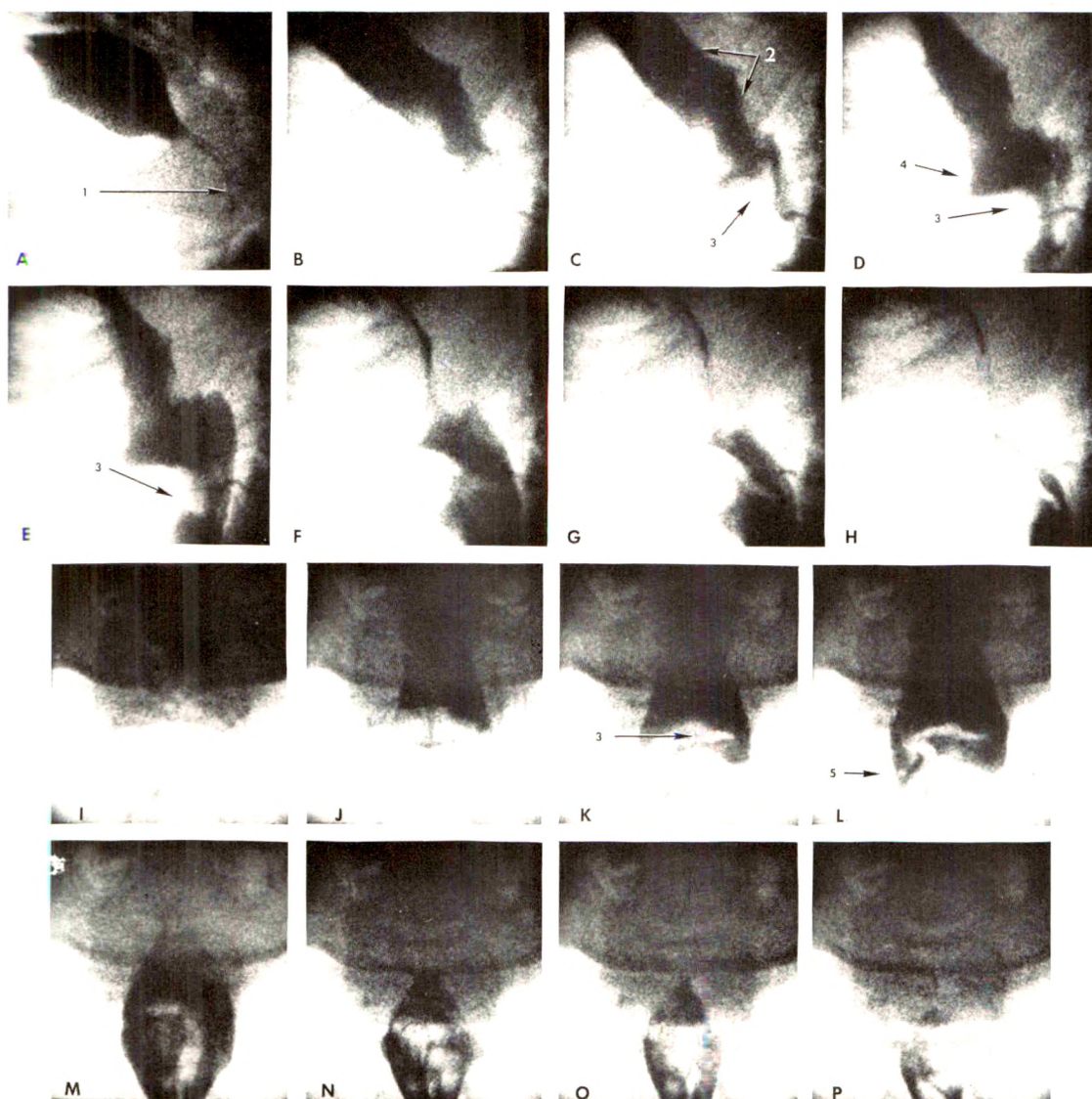


FIG. 3. Normal barium swallow studies (oropharyngeal stage). Note: Symmetric pharyngeal constriction and absence of pharyngeal retention. Selection of cine-frames in lateral (A-H) and posteroanterior (I-P) projection. 1—posterior surface of the tongue; 2—soft palate; 3—epiglottis; 4—valleculae; and 5—piriform sinus.

tion of muscular activity often replaces the rapid motion seen normally. Complete disorganization of the swallowing sequence represents the most severe type of motility disturbance. The participation of muscles and organs usually not employed in the act of swallowing frequently discloses pathologic change in pharyngeal motility. For example, movements of the head and jaws may be present to aid the transport of food to the esophagus. These movements are

usually accompanied by additional manifestations of abnormal deglutition, including stasis and aspiration.

2. *Residual filling of the pharyngeal recesses* occurs because of altered sensitivity of adjacent mucosa, decreased muscular tone, mechanical obstruction, changes in recess shape and neuromuscular disturbance. Atrophy of fat tissue, normally found below the valleculae and supporting the epiglottis, may be responsible for the dila-

tation of these pouches and subsequent retention of food (barium). This is presumably the basis for this observation in cachectic patients. Residual filling of pharyngeal recesses in elderly patients can also be explained by the loss of elastic tissue in the pharynx. The use of pasty barium may produce prolonged filling of the valleculae in normal individuals. Conversely, residual filling of the pharyngeal recesses is invariably present in patients with peripheral or central motor nerve paralysis. Thus, vallecular retention, frequently associated with a swallowing disorder, is valuable as a differential sign only if additional roentgenographic changes are observed.

3. *Pharyngeal stasis* occurs when pathologic outpouching of the pharynx (lateral pharyngeal pouches, diverticula) or dilatation of the pharyngeal cavity is present. Stasis is a reliable sign of pharyngeal motor disturbance and is seen in pharyngeal paralysis or in mechanical obstruction produced by intrinsic or extrinsic tumors.

4. *Misdirected swallowing* (aspiration) is one of the most striking abnormalities observed clinically as well as roentgenographically. The bolus deviates from its normal pathway and proceeds either into the nasal cavity or trachea. This phenomenon occurs when there is impairment of the closing mechanism or pathologic communication to the nasopharynx and trachea. Retained pharyngeal contents may be aspirated during respiration without swallowing. Uni- or bilateral paresis of the soft palate, morphologic defects of the hard palate or tumors of the epipharynx will lead to reflux into the nasopharynx. Paralysis of muscles of the floor of the mouth and interference with the function of the recurrent laryngeal nerve also result in suppression of important safety mechanisms. Examples of interference with motor function are seen in myasthenia gravis, and postresectional alterations of pharyngeal anatomy and innervation; and examples of interference with sensory protection are peripheral toxic injuries (e.g., diphtheria) or anesthesia of the pharynx.

Abnormal deglutition associated with *neuromuscular disorders* may be observed in the following categories:

1. Disorders involving the brainstem: posterior inferior cerebellar artery syndrome; poliomyelitis; amyotrophic lateral sclerosis; multiple sclerosis; and syringomyelia.

2. Myoneural junction disorders such as myasthenia gravis.

3. Disorders of muscle itself including the muscular dystrophies, ocular myopathy, sarcoidosis, dermatomyositis and systemic lupus.

4. Lesions of undetermined pathogenesis: pharyngoesophageal incoordination;^{6,7} sideropenic dysphagia.

EXPERIMENTAL PROCEDURE

The cinefluorographic apparatus employed for these studies consisted of a 9 inch image amplifier, an image orthicon television system and two 16 mm. motion picture cameras. One camera recorded the fluoroscopic image displayed on a television monitor; the other, the images appearing on the output phosphor of the image amplifier. Monitor recording was used when detail was relatively unimportant and when amplifier recording was likely to require undesirably high radiation dosage levels. Roentgen rays were provided by a grid controlled roentgenographic tube. This tube restricts the delivery of radiation to the time when the shutters of the motion picture cameras are open, thereby maintaining patient dosage at minimal levels. The roentgen-ray tube was also equipped with a small (.5 mm.) focal spot to ensure images of high resolution. Cine-recordings were made at a film speed of 30 frames/second.

Cinefluorographic examinations of pharyngeal deglutition were carried out in prone and erect positions. On lying prone, patients with "high dysphagia" usually had more difficulty swallowing than in the erect position. However, roentgenographic abnormalities such as webs, cricopharynx-

geal sphincter defects and motor weakness of pharyngeal contraction, are exaggerated in this position and, therefore, easier to detect. Cine-recordings were obtained in the lateral projection with the patient lying on his right side. They included the oropharyngeal stage of swallowing and the transport of the bolus through the cricopharynx into the upper esophagus. With the patient standing erect, barium swallow studies were made in the posteroanterior and lateral projections. This part of the examination provides valuable information concerning tone of the pharynx and pooling of contrast material in the pharyngeal recesses. In addition, the posteroanterior projection is useful in paralysis of specific groups of muscles to demonstrate asymmetric contraction of the constrictors and deflection of the bolus to either food channel as it passes through the pharynx.

Conventional barium suspension was given unless the patient's history strongly suggested aspiration. Under these conditions gastrografin is of equal value. Studies of pharyngeal deglutition were never begun with barium paste. A patient with paralysis of the constrictor muscles, unable to force the material into the esophagus, may obstruct his airway, leading to considerable respiratory distress.

REPRESENTATIVE EXAMPLES

A representative example of a patient with a brainstem lesion, namely *thrombosis of the posterior inferior cerebellar artery*, is shown in Figure 4. Normally, in response to swallowing, pharyngeal contraction occurs and the cricopharyngeal sphincter relaxes. Sphincter relaxation begins prior to pharyngeal contraction above it. In this patient decreased tone and dilatation of the pharynx with inadequate relaxation of the cricopharyngeal sphincter was observed, producing a deformity of the pharynx which was interpreted as a tumor mass on conventional roentgenograms (Fig. 4). Cinerentgenographically, however, no persistent deformity was present; the walls were pliable (Fig. 5, A-D). This was simul-



FIG. 4. Brainstem lesion: posterior inferior cerebellar artery syndrome. Spot roentgenogram obtained during fluoroscopic examination. Provisional diagnosis at this time: hypopharyngeal tumor. Note: Aspiration into laryngeal vestibule and trachea, contracted cricopharyngeal sphincter, barium retention and stasis in pharyngeal recesses and cavity.

taneously demonstrated on the manometric record.

Disorders involving the myoneural junction, e.g., *myasthenia gravis*, the pharynx, the cricopharyngeus and upper esophagus, demonstrate motor abnormalities (Fig. 6, A-L). Hesitancy in initiating deglutition with inefficiency in forming a bolus on the back of the tongue results in a measurable delay in cleansing the mouth and oral pharynx. Movements of the tongue are slow and inefficient and become progressively weaker and slower as continued attempts to swallow are made. This easy fatigability of the pharyngeal motor activity is a characteristic feature of myasthenia gravis. During the pharyngeal stage, prolonged coating

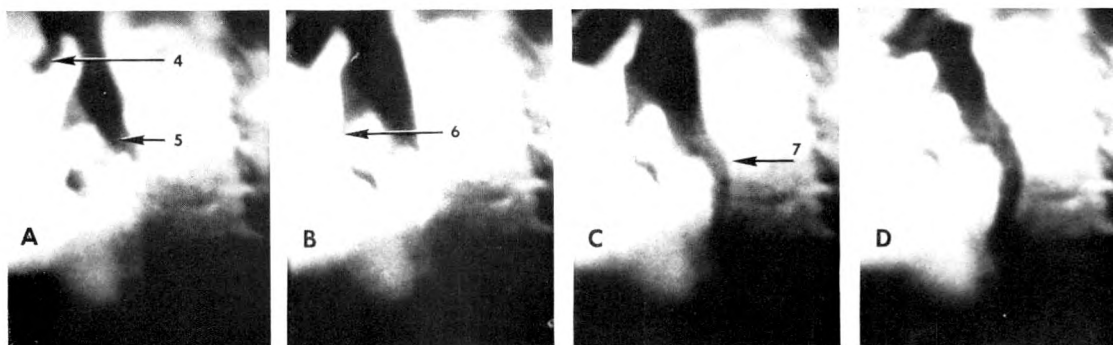


FIG. 5. (A-D) Same patient as in Figure 4. Note: Retention of barium in valleculae (4) and piriform sinuses (5), elongated incompletely relaxed cricopharyngeal sphincter (7), in spite of deglutition and maximal distention of the pharynx, aspiration into laryngeal vestibule (6).

of the walls and pooling of barium in the pharyngeal recesses are associated with loss of tone and ballooning of the pharyngeal cavity. Nasal regurgitation may occur during successive swallows due to a diminishing ability to raise the pharynx, to close the epipharynx and to approximate the posterior wall and palate effectively to the base of the tongue. In the posteroanterior projection, passage of the bolus through the pharynx is symmetrical by virtue of the generalized muscle involvement. Never is there any spasm or hypertrophy of the cricopharyngeal sphincter as is seen in brainstem lesions, sideropenic dysphagia or pharyngoesophageal incoordination. The subcutaneous injection of prostigmine has

helped to establish the diagnosis of myasthenia with principal involvement of the muscles of deglutition.⁵ By documenting and quantitating improvement with prostigmine, it has helped ascertain the adequacy of therapy by the return of motor function toward normal. The manometric and symptomatic improvement after medication correlates well with the improved flow through the cricopharyngeus observed by cinerentgenography though completely normal function is often not restored.

The collagen diseases are among the disorders involving the striated muscles of the pharynx. Dermatomyositis characteristically produces pharyngeal dysphagia whenever the upper gastrointestinal tract is

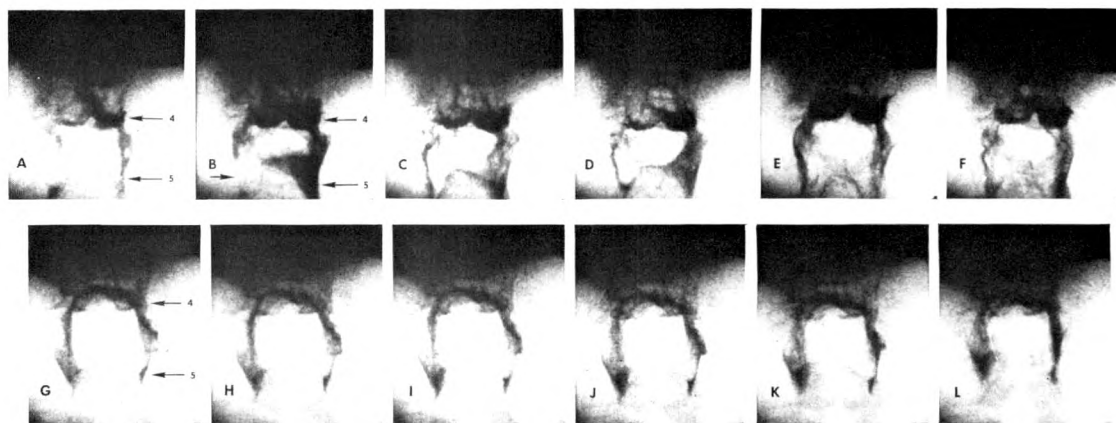


FIG. 6. Myasthenia gravis. Barium swallow studies before (frames A-F) and 30 minutes following the subcutaneous injection of 1 mg. of prostigmine (frames G-L). Note: Persistent retention of barium in valleculae (4) and piriform sinuses (5), moderate distention of pharyngeal cavity (A-F), improved deglutition, small amount of retained barium in pharyngeal recesses (G-L).

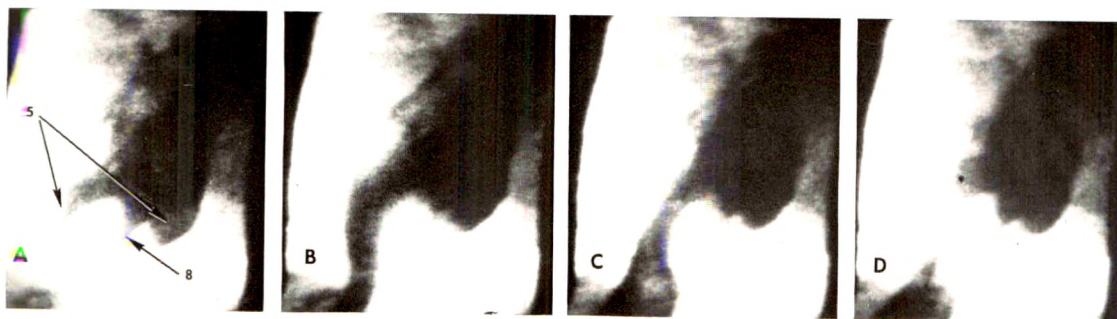


FIG. 7. (A-D) Acute systemic lupus erythematosus. Barium swallow studies in recumbent position, kinescopic recording. Note: Pharyngeal paralysis with stasis of barium, residual weak activity of pharyngeal musculature on the left side. Barium proceeds through right food channel, aspiration into laryngeal ventricle (8). Piriform sinuses (5).

involved. The patient in Figure 7, A-D, presents an example of acute systemic lupus erythematosus affecting the pharyngeal constrictors to the degree of complete paralysis. In addition to motility disturbances and residual filling of recesses, stasis and aspiration could be observed. Hence, in this instance, all 4 of the roentgenographic signs

of pathologic deglutition have developed. Another, less frequent disorder involving the striated muscle portion of the pharynx, cricopharyngeus and esophagus, and occasionally the smooth muscle portion of the esophagus is myotonia dystrophica (Fig. 8, A-K). Motor disturbances in myotonia dystrophica can be produced or exagger-

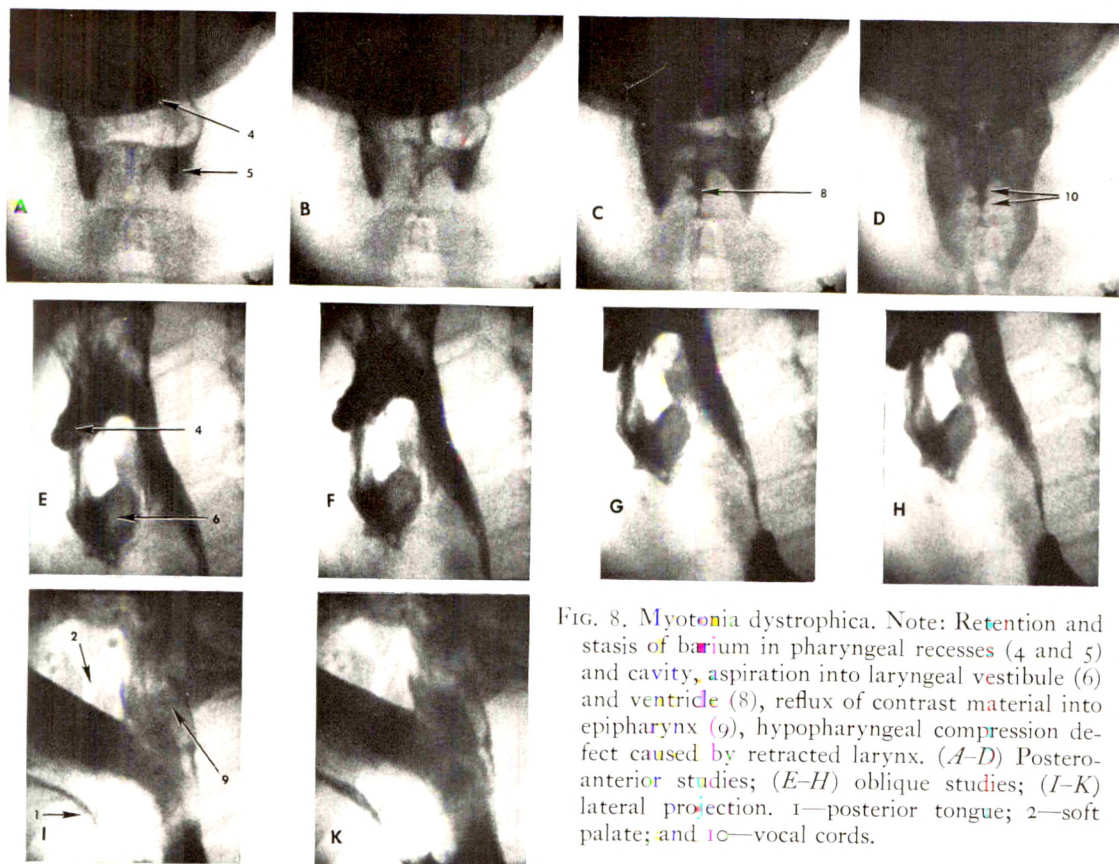


FIG. 8. Myotonia dystrophica. Note: Retention and stasis of barium in pharyngeal recesses (4 and 5) and cavity, aspiration into laryngeal vestibule (6) and ventricle (8), reflux of contrast material into epipharynx (9), hypopharyngeal compression defect caused by retracted larynx. (A-D) Postero-anterior studies; (E-H) oblique studies; (I-K) lateral projection. 1—posterior tongue; 2—soft palate; and 10—vocal cords.

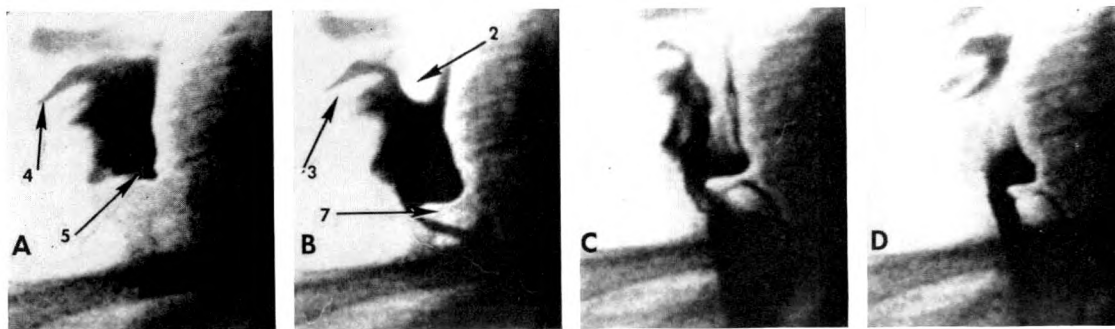


FIG. 9. (A-D) Pharyngoesophageal incoordination. Partial obstruction caused by contracted hypertrophic cricopharyngeal sphincter (7). Film sequence obtained in lateral projection. 2—soft palate; 3—epiglottis; 4—valleculae; and 5—piriform sinuses.

ated by cold liquids.⁶ Successive swallows frequently show improvement in pharyngeal contractions and decreased duration. The disease process in the pharynx may have progressed to the degree that complete paralysis with nasal and tracheal aspiration occurs. The absence of incomplete cricopharyngeal relaxation, the improvement of pharyngeal contraction during successive swallows and, conversely, the exaggeration of abnormal pharyngeal motility produced

by swallowing ice water are helpful and distinguishing roentgenographic signs.

Pharyngoesophageal incoordination and sideropenic dysphagia represent two disorders with "high dysphagia" in which the pathogenesis is undefined. The patient in Figure 9, A-D, demonstrates hypertrophy and spasm of the cricopharyngeal sphincter with resultant incomplete obstruction of the flow of barium associated with pharyngeal dilatation. The function of this sphinc-

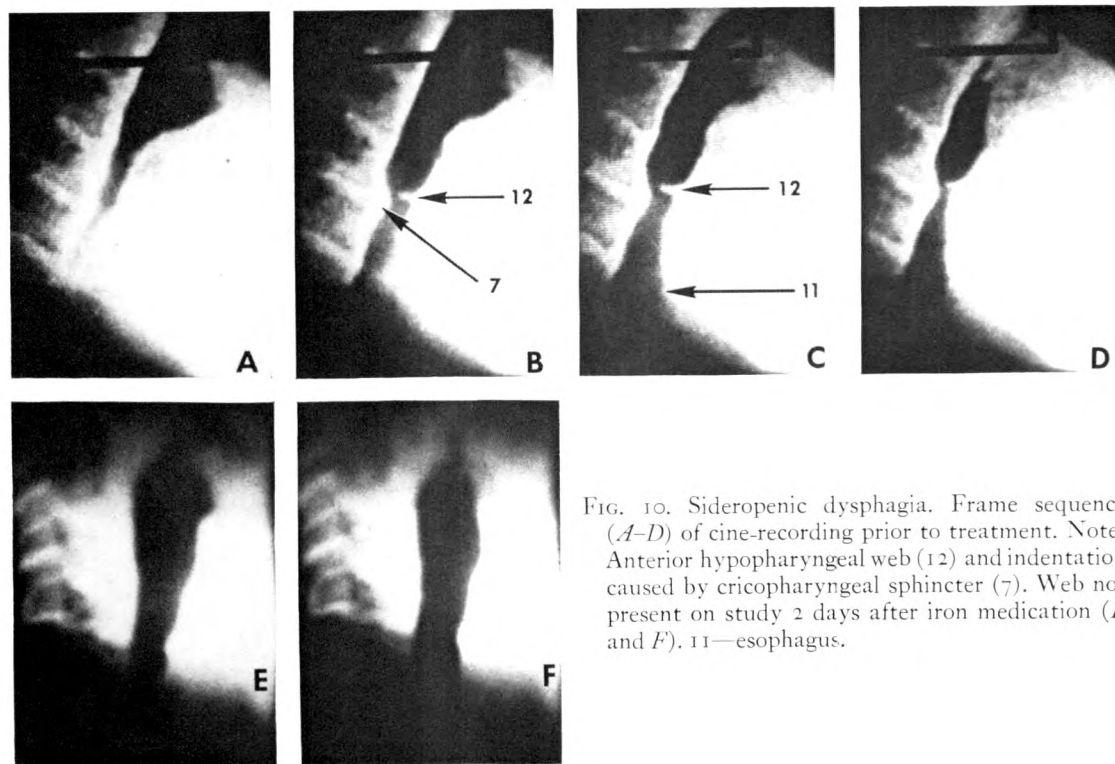


FIG. 10. Sideropenic dysphagia. Frame sequence (A-D) of cine-recording prior to treatment. Note: Anterior hypopharyngeal web (12) and indentation caused by cricopharyngeal sphincter (7). Web not present on study 2 days after iron medication (E and F). 11—esophagus.

ter is deranged similar to functional disturbances of other sphincters of the gastrointestinal tract. This incomplete relaxation of the cricopharyngeal sphincter produces indentation of the barium column as it passes through the postcricoid region. A longitudinal myotomy of the cricopharyngeus and the lower portion of the inferior pharyngeal constrictor was performed in order to relieve obstruction. Postoperatively, the dysphagia diminished considerably and cinerontgenographic studies showed easy passage of the barium through the cricopharyngeal region. However, contraction of the pharyngeal constrictors is still inadequate.

Atrophy and morphologic changes of the pharyngeal mucosa may be responsible for the dysphagia as seen in *sideropenic disturbances of deglutition* (Fig. 10, A-F). Prior to treatment with iron compounds, an anterior web frequently can be seen indenting the anterior wall of the hypopharynx, usually close to the location of the cricopharyngeal sphincter. The web is pointed, semicircular and occasionally produces obstruction. In addition, a posterior indentation at the level of the sixth cervical vertebra indicates spasm of the cricopharyngeal sphincter. Within hours following the application of iron medication, both the web and the spasm of the upper esophageal sphincter may disappear.

SUMMARY

Pharyngeal dilatation may be produced by obstructing lesions of the hypopharynx and upper esophagus, or it may be associ-

ated with neurologic and muscle diseases. Mechanical causes for pharyngeal dysphagia can usually be recognized, but characteristic roentgenologic manifestations of neuromuscular lesions have been more difficult to identify.

Cineroentgenography, combined with simultaneous intraluminal pressure recordings, has established distinguishing combinations of roentgenographic signs in disorders involving the brainstem, disorders producing interference at the myoneural junction and disorders of the muscle itself.

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ALLISON AND JOHNSTONE'S ANOMALY*

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EPONYMOUS titles are not popular, but alternative names for Allison and Johnstone's anomaly are cumbersome. It has been called "oesophagus lined with gastric mucosa" and "gullet lined with columnar epithelium." It is not inappropriate that the names of Professors Philip Allison and A. S. Johnstone should be associated with it, since they were the first to describe this fascinating condition.

It is only during the last 10 to 20 years that our attention has been directed to the problem of hiatus hernia, and it is not surprising that much is still unknown on the subject and its related abnormalities. The association between hiatus hernia and reflux, on the one hand, and peptic esophagitis and stricture, on the other, was reported by Allison¹ in 1948.

Barrett,² in 1950, drew attention to the confusion which had arisen over the term "peptic ulcer of the oesophagus," and showed beyond doubt that endoscopists and pathologists had been talking about two different things. The endoscopists were describing the appearances produced by the digestive action of acid pepsin on the squamous epithelium of the esophagus, which is now known as reflux esophagitis. The ulcer so produced was little more than a superficial desquamation, being large in area but without depth, and although obvious to the endoscopist it became insignificant after death. The pathologists, on the other hand, were describing a deep penetrating ulcer of the esophagus, which was brought to their notice by a sudden lethal hemorrhage.

In 1942, Dick and Hurst³ noted that penetrating ulceration of the gullet was also associated with hiatus hernia, but because of postmortem autolysis they failed to note that the adjacent mucosa was gas-

tric, and thus came to the erroneous conclusion that the deep ulcer was the result of reflex esophagitis. It remained for Barrett² in 1950 to point out that the deep penetrating ulcers always occurred in gastric mucosa, and because of this the condition of deep penetrating ulcer of the gullet is associated with his name. It is unfortunate that at this time he drew the wrong conclusion that since this ulcer was in gastric mucosa it was therefore in hiatus hernia.

The true state of affairs was not revealed until 1953, when Allison and Johnstone² described 8 cases of hiatus hernia in which a portion of adjacent gullet was lined with "gastric mucosa." They called this abnormal segment gullet, in spite of its gastric lining, because: (1) it had no peritoneum in relation to it; (2) it had an esophageal blood supply; and (3) it appeared, externally, identical to normal esophagus, and even the "gastric mucosa" lining it had some esophageal features. These were deep esophageal mucous glands, which are a feature of gullet and not of stomach, and also, at any level in this prefixed "gastric mucosa" islets of squamous epithelium could be found.

It therefore becomes clear on reviewing Barrett's cases that Barrett's ulcer is a deep penetrating ulcer of this abnormally lined gullet. Barrett continued for years to argue that it was more proper to term this abnormal segment stomach, until 1958 when he published an article entitled "Lower Gullet Lined with Columnar Epithelium," finally capitulating to the strength of the argument put forward by Allison and Johnstone. The difference between the two ulcers as to frequency, site, pathology, roentgen signs and presentation is shown in Table 1.

* Extracted from a thesis which is the property of the University of Edinburgh.

TABLE I
COMPARISON OF THE TWO TYPES OF SIMPLE ULCERATION OF THE GULLET

	Ulcer of Peptic Esophagitis	Barrett's Ulcer
Frequency	Common	Rare
Site	In squamous epithelium at the mucosal transition	In gastric mucosa anywhere in gullet
Pathology	Superficial desquamation only	Deep penetrating ulcer
Roentgenologic Manifestations	Stricture only	Ulcer niche and barium rest
Presentation	Dysphagia	Severe, often lethal, hemorrhage

ANATOMIC CONSIDERATIONS

In order to understand hiatus hernia and to relate roentgenology to surgery, certain anatomic relations have to be defined. It soon became apparent that endoscopic levels measured from the upper alveolus and related to other mediastinal structures were not the same as those measured by anatomists on the cadaver. This is because the endoscope, no matter how skillfully inserted, stretches the gullet and thus exaggerates distance. It is noteworthy that an endoscopist may make a lesion appear lower than it is but never higher. By comparing the endoscopic distances of structures from the upper alveolus with their level apparent on the roentgenogram, it was possible to establish a more realistic relationship. These levels which are shown in Figure 1 are:

Upper end of the esophagus—	15 cm.
Upper border of aortic arch—	20 cm.
Lower border of aortic arch—	23 cm.
Left main bronchus —	26 cm.
Inferior pulmonary vein —	30 cm.
Hiatus esophageus —	38 cm.

The blood supply of the thoracic portion of the esophagus is by segmental branches directly from the aorta, which run transversely or downwards, the lowest one coming off at the level of the lower pulmonary vein. The esophagus in the region of the hiatus esophageus is supplied by a small twig from the left gastric artery. Perito-

neum is reflected from the stomach around the hiatus esophageus, save for a small area posteriorly—the bare area of the stomach. All these features are shown in Figure 1.

It is important to find a suitable definition for esophagus or gullet, because if it be that portion of the upper alimentary tract lined with squamous epithelium, then there are cases which could be said to have 2 stomachs and hardly any esophagus. If, on the other hand, it is the tubular portion of the upper alimentary tract joining pharynx to stomach, then the lower 2 cm. of the tube is normally lined with "gastric mucosa,"¹² the lower end point being where the tube widens abruptly into the stomach. For the purpose of this paper, the latter definition has been accepted.

In Figure 2 the anatomic relationships of a straight forward short esophagus type hiatus hernia are shown with the endoscopic transition at 34 cm., corresponding to its anticipated roentgenographic site. The gastric dome is supplied by the left gastric artery, and it has a peritoneal sac situated anterolaterally. The adjacent esophagus, which is lined with squamous epithelium, is supplied by the lowest segmental branch of the aorta running downwards.

PHYSIOLOGIC CONSIDERATIONS

The intermittent constriction of the cardiac sphincter mechanism is a useful landmark to mark the lower end of the esophagus. Although it may not be exactly at the

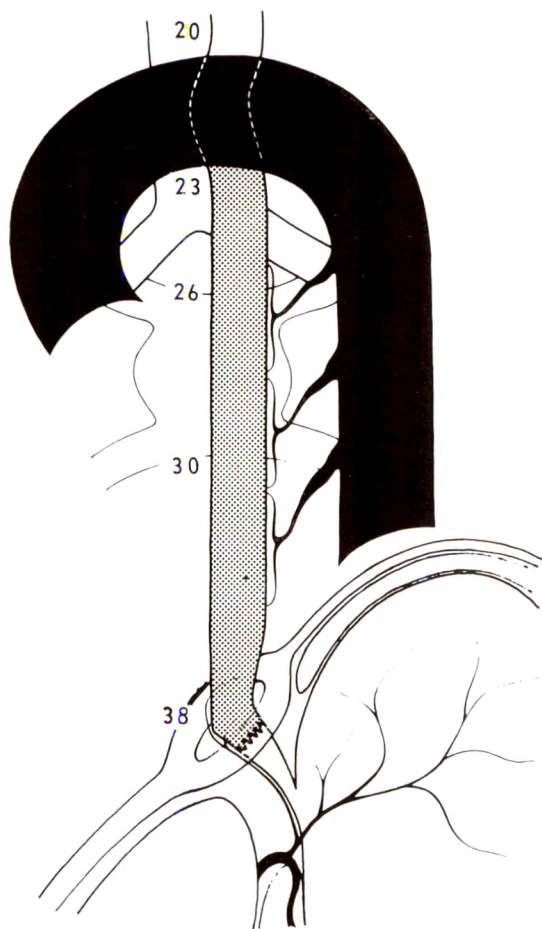


FIG. 1. The mediastinal relations of the thoracic esophagus are shown along with the endoscopic levels of these structures measured in centimeters from the upper alveolus. The shaded area indicates squamous epithelium and the unshaded area gastric mucosa. It will be noticed that the lower 2 cm. of gullet has the same epithelium as the stomach. The blood supply of stomach and gullet is indicated along with the peritoneal reflection between stomach and diaphragm.

lower end of the gullet, it is at least within 2 cm. of it. It is apparent to the radiologist as a localized narrowing intermittently present at the lower end of the gullet. There have been various reports of an anatomic sphincter¹⁴ and there have been at least as many denying its existence.^{5,17}

Regardless of the difficulty in demonstrating an anatomic sphincter, there is no doubt as to the presence of a physiologic one. Further confirmation of the existence

of a physiologic sphincter mechanism is supplied by the technique of intraluminal esophageal pressure recordings. A high pressure zone was demonstrated by Fyke, Code and Schlegel¹⁰ in 1956 similar to that encountered at the pharyngoesophageal junction, and it extended from 1 to 2 cm. above the hiatus to 1 to 2 cm. below it. These findings were confirmed by Hightower¹³ in 1958. It would appear that there is a physiologic sphincter mechanism, and it is normally closed unless compelled to

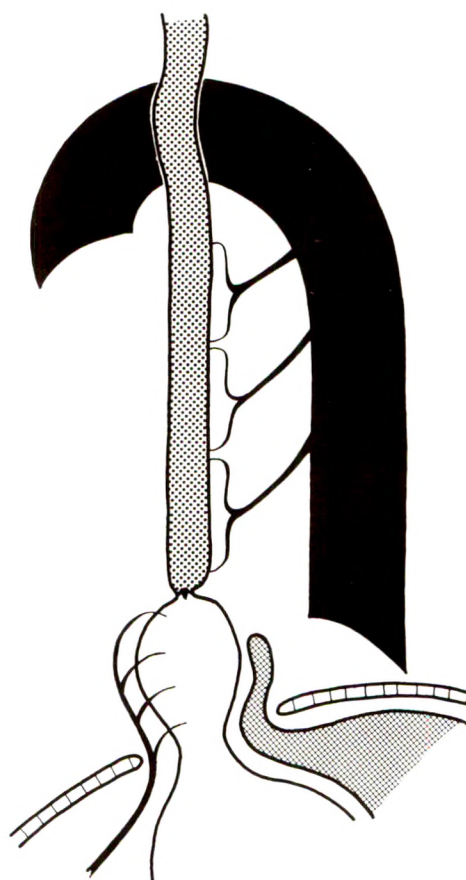


FIG. 2. Showing the anatomy of a typical short esophagus type hiatus hernia. The blood supply of herniated stomach from the left gastric artery and the small anterolateral peritoneal sac are indicated. The mucosal transition is at the junction between the shaded and nonshaded areas in the anticipated site. The adjacent esophagus, which is lined with squamous epithelium, is supplied by the lowest segmental branch from the aorta running downwards.

open in advance of a peristaltic wave. Any doubt as to whether this high pressure zone was due to any external mechanism has been removed by the fact that this high pressure zone is still present, but at a higher level, when associated with hiatus hernia. This has been shown by Carré and Astley⁶ in 1958 in the United Kingdom, and by Texter, Lazar, Puletti and Vantrappen²¹ in 1959 in the United States, and it is seen roentgenoscopically as an intermittent constriction between the esophagus and the hernia.

MATERIAL AND METHOD

To define the incidence of this condition, 100 cases filed in the disease index as hiatus hernia and operated upon were carefully reassessed. It should be stressed that in this series all roentgenologic phenomena were exactly correlated with the findings of the endoscopist, the surgeon and the pathologist, and it is only in this way that any meaningful roentgenologic observations can be established, and the creation of a new mythology avoided.

Eighty-seven per cent of the cases appeared to fit the picture of a simple hiatus hernia, but in 13 per cent it was obvious that a portion of the gullet was lined with gastric mucosa. This condition was made the subject of a special study, and other cases were added to a total of 27.

ILLUSTRATIVE CASES

CASE 1. In this 53 year old male, three years previously a diagnosis of short esophagus type hiatus hernia had been made at another hospital, and he had been advised to sit up at night. Six weeks before admission he sustained a back injury which was diagnosed as a slipped disk and he was confined to bed to lie flat on boards. One week later, at night while lying flat on his back, he experienced severe retrosternal discomfort and collapsed. A diagnosis of coronary thrombosis was made. He was admitted to the hospital where he later vomited half a pint of dark blood not mixed with food. He vomited blood again on two subsequent occasions. Barium meal examination (Fig. 3A) showed a

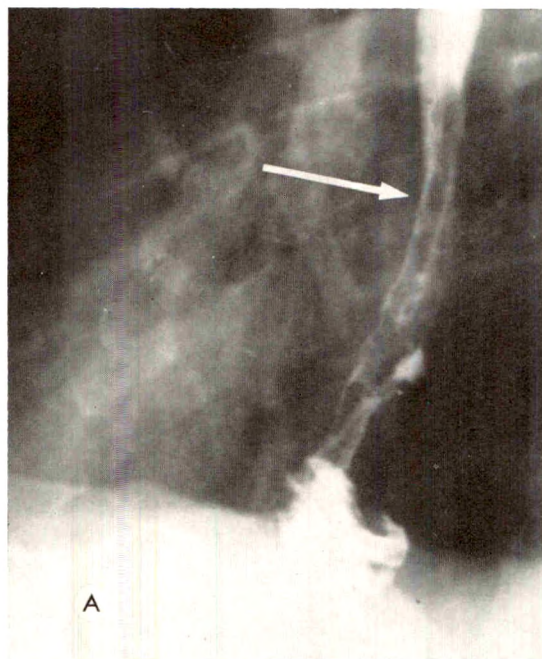


FIG. 3. Case 1. (A) Barium meal examination showing a moderate sized hiatus hernia with an ulcer crater well above the junction of esophagus and stomach. The mucosal transition was, in fact, well above the ulcer at the site indicated by the arrow. There is no roentgenoscopic change at this level.

moderate sized hiatus hernia of the short esophagus type with regurgitation. There was an ulcer crater present above the estimated position of the cardiac orifice. Endoscopic examination showed at 27 cm. a normal transition from esophageal to gastric mucosa. At 33 cm. there was an ulcer with a white base 1 cm. in diameter. It had a clean cut edge and appeared to be situated posteriorly in the gastric mucosa. Tissue was taken (1) from the edge of the ulcer, (2) from the mucosa at the level of the ulcer and (3) from the mucosa proximal to the ulcer. The pathologic report on these 3 specimens was that the mucosa was of the type normally found in the cardia of the stomach.

At operation (standard left thoracotomy) there was no obvious gastric dome in the pulmonary ligament. The hiatus admitted 5 fingers. On closer examination it was apparent that there was an inflammatory lesion in relation to the distal esophagus, and it was judged that an ulcer was penetrating the pulmonary ligament. The esophagus was mobilized and found to be large and thick walled, and a site

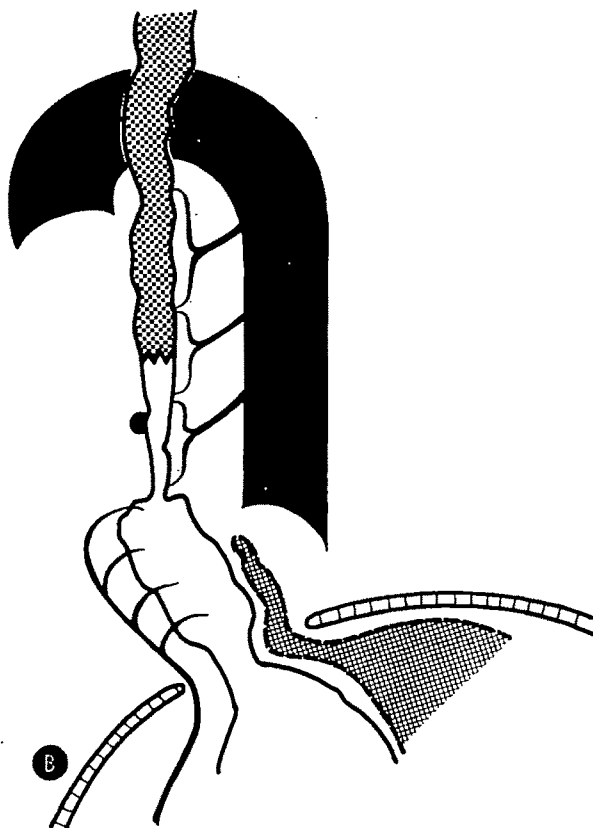


FIG. 3. (B) Diagram showing the pathologic anatomy as determined by biopsy and at operation followed by examination of the resected specimen. Shaded area indicates squamous epithelium and nonshaded area gastric mucosa. Cross hatching is peritoneum.

for section was chosen at the crossing of the left main bronchus. The stomach was divided and an end to end esophagogastrostomy was performed. The pathologic report on the ulcer was that the changes were those of chronic gastric ulceration.

Comment. The transition of squamous epithelium to gastric mucosa was shown at endoscopy to be at 27 cm. (at the level of the left main bronchus), and this was confirmed by biopsy. There was no roentgenologic evidence of any change at this level. The presence of a large hiatus supports the diagnosis of hiatus hernia, although self-reducing herniations are probably rare. The ulcer niche represents a Barrett's ulcer in a segment of esophagus lined with gas-

tric mucosa. The salient features of the endoscopic, operative and pathologic findings in relation to the roentgenographic appearance are shown in Figure 3B.

CASE II. A female of 51 years of age complained of dysphagia for 6 months. Barium meal examination (Fig. 4A) showed a short esophagus type hiatus hernia with free gastroesophageal reflux. There was a stricture at the level of the aortic arch, and a local view (Fig. 4B) showed an ulcer below the stricture. The mucosa below the stricture appeared even smoother than the mucosa above it. Endoscopic examination showed typical changes of peptic esophagitis, and stricture at 23 cm. (lower border of the aortic arch). Biopsy just distal to the stricture revealed gastric mucosa (Fig. 4C) and a few oxyntic cells were demonstrated by special stain. The patient died of a massive hemorrhage and at postmortem examination the gullet below the stricture was found to be lined with gastric mucosa. In this gastric mucosa there was a deep penetrating ulcer eroding a large vessel in its base. (The preceding features are related to the roentgenographic appearance in Figure 4D.)

Comment. There was a large portion of the gullet between the hernia and the stricture lined with "gastric mucosa," and the deep ulcer in this segment was a Barrett's ulcer. The gastric mucosa lining the gullet here had no gastric features roentgenologically, and indeed appeared smoother than the squamous epithelium above.

CASE III. A female, aged 60 years, complained of regurgitation for 20 years, and dysphagia for 5 years. At barium meal examination (Fig. 5, A and B) a short esophagus type hiatus hernia with free gastroesophageal reflux was found. There was a stricture, purse-string in type, well above the aortic arch, producing some proximal stasis and dilatation. Between the stricture and the lower end of the gullet, marked by the cardiac sphincter mechanism, lay the major portion of the gullet. Endoscopically, there were changes of peptic esophagitis leading to a stricture at 20 cm. (upper border of the aortic arch). Biopsy at 27 cm. showed gastric mucosa with deep esophageal mucous glands beneath (Fig. 5C). At operation, a small hiatus hernia with peritoneal sac in relation to it was found,

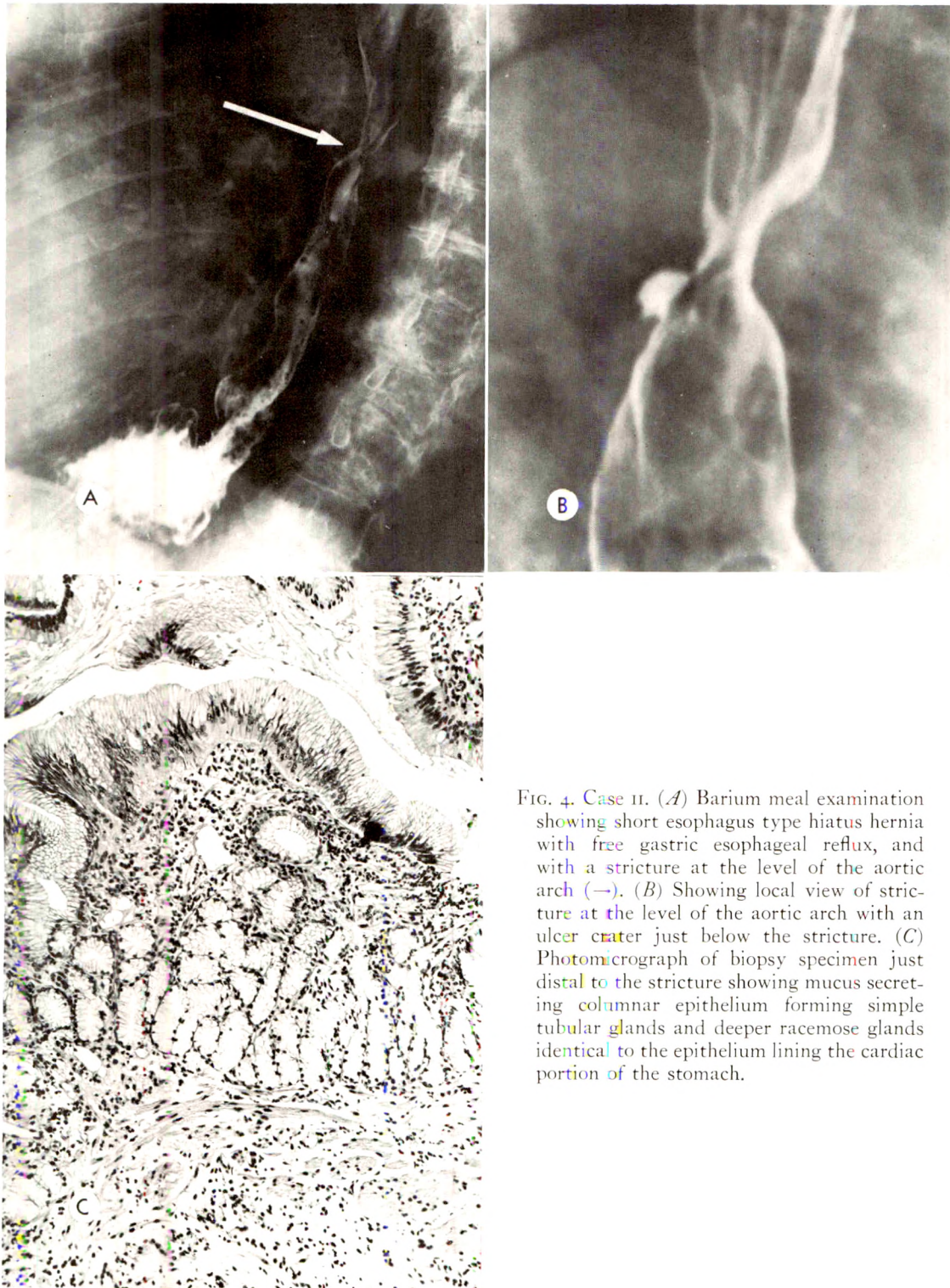


FIG. 4. Case II. (A) Barium meal examination showing short esophagus type hiatus hernia with free gastric esophageal reflux, and with a stricture at the level of the aortic arch ($\rightarrow$). (B) Showing local view of stricture at the level of the aortic arch with an ulcer crater just below the stricture. (C) Photomicrograph of biopsy specimen just distal to the stricture showing mucus secreting columnar epithelium forming simple tubular glands and deeper racemose glands identical to the epithelium lining the cardiac portion of the stomach.



FIG. 4. (D) The features of this case as determined by biopsy and postmortem examination are shown diagrammatically in relation to the roentgenographic appearances. The stricture is situated too low in the diagram. The shaded area represents squamous epithelium and the unshaded area gastric mucosa.

and proximal to the hernia was normal esophagus. The hernia was reduced and the hiatus narrowed. (The preceding features are related

to the roentgenographic appearance in Figure 5D.)

Comment. The fact that most of the gullet was lined with gastric mucosa was not apparent roentgenologically. Indeed, this abnormal mucosa had all the appearances of squamous epithelium. The repair of the hernia, with cessation of reflux, cured the patient of dysphagia and caused healing of the endoscopically manifest peptic esophagitis which resulted in considerable improvement in the roentgenologic features of the stricture.

Adenocarcinoma. This has been reported as a complication of Allison and Johnstone's anomaly in pathologic specimens. In an attempt to see if this diagnosis could be made in life, 136 cases of adenocarcinoma were reviewed. The condensed data from this review are shown in Table II. The findings are of interest in relation to a review of adenocarcinoma of the gullet by Le Roux¹⁸ in 1961. Conveniently, Le Roux, working in the same unit, reviewed the same cases, and in his cases of adenocarcinoma occurring at any level all were continuous with gastric mucosa inferiorly, and none had squamous epithelium below. All these adenocarcinoma cases were, therefore, derived from gastric mucosa and none from esophageal mucous glands.

In view of the disruption of normal anatomy produced by carcinoma and the ability of these tumors to grow upwards for a considerable extent, it was impossible, in retrospect, to be certain that they derived

TABLE II
ENDOSCOPIC LEVELS OF 176 CASES OF ADENOCARCINOMA

Endoscopic Levels	Over 40 cm.	35-40 cm.	30-35 cm.	22-30 cm.
No. of Cases	66	56	37	17
Percentage	37.5%	32%	20.5%	10%
Associated Hernias	2 para-esophageal hernias	No hernias	2 hernias (short esophagus type)	7 hernias (short esophagus type)

* Note: The upper level of the tumor seen endoscopically was measured from the upper alveolus.

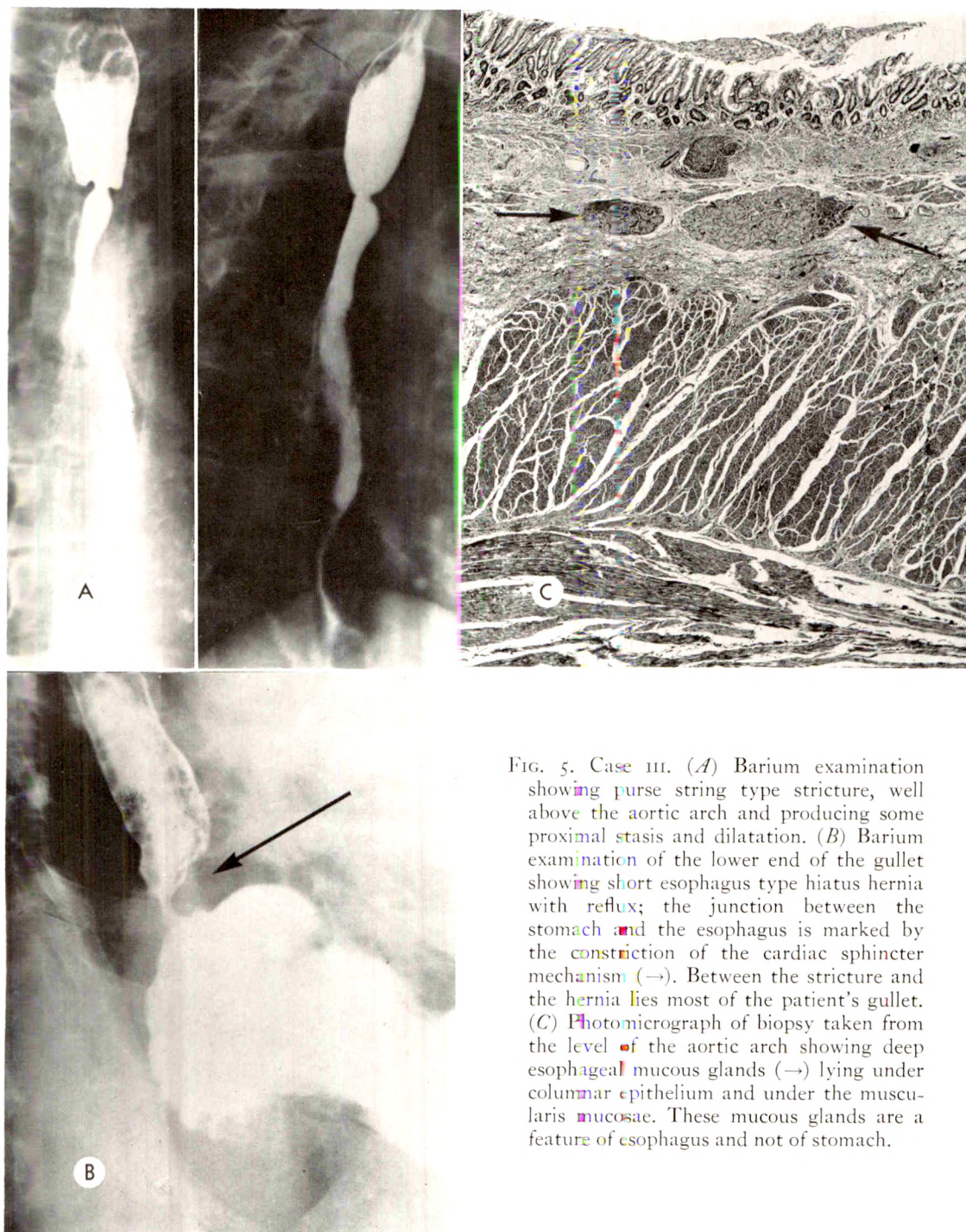


FIG. 5. Case III. (A) Barium examination showing purse string type stricture, well above the aortic arch and producing some proximal stasis and dilatation. (B) Barium examination of the lower end of the gullet showing short esophagus type hiatus hernia with reflux; the junction between the stomach and the esophagus is marked by the constriction of the cardiac sphincter mechanism (→). Between the stricture and the hernia lies most of the patient's gullet. (C) Photomicrograph of biopsy taken from the level of the aortic arch showing deep esophageal mucous glands (→) lying under columnar epithelium and under the muscularis mucosae. These mucous glands are a feature of esophagus and not of stomach.

from gastric mucosa in gullet in more than 2 cases, since in only 2 cases was the mucosal transition above the tumor. An example of this is given in the following case.

CASE IV. A female, aged 71 years, complained of dysphagia for 1 year. Barium meal examination revealed a short esophagus type hiatus hernia with reflux, and above the hernia was an

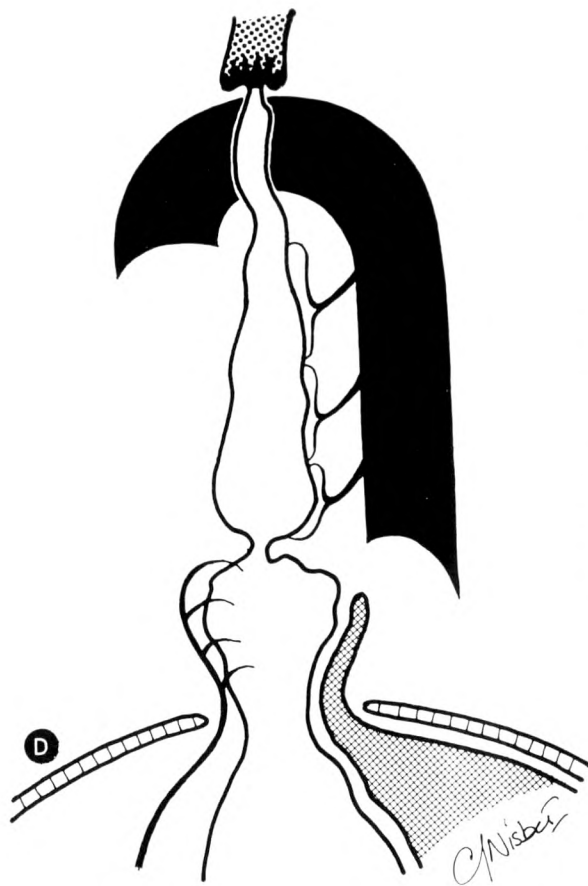


FIG. 5. (D) The features assessed by biopsy and at operation are shown diagrammatically in relation to the roentgenographic appearances. The shaded area indicates squamous epithelium and the unshaded area gastric mucosa.

obvious tumor obstructing the gullet (Fig. 6). Endoscopy at 21 cm. (upper border of the aortic arch) showed a normal transition from squamous to gastric mucosa, and at 30 cm. there was tumor. Biopsy proximal to the tumor showed gastric mucosa and biopsy of the tumor showed adenocarcinoma. At operation, the presence of a hiatus hernia with a tumor of the gullet above was confirmed but the case was inoperable.

Comment. The high transition was not apparent roentgenologically and adenocarcinoma is, therefore, one complication which does not help towards a roentgenologic diagnosis of Allison and Johnstone's anomaly.

HISTOLOGIC FEATURES

The histologic features of the prefixed gastric mucosa have been assessed from the routine biopsy material obtained at endoscopy, or from the microscopic examination of resected specimens. From these random samples, it appears that in 18 of the cases, the gullet was lined with mucus secreting columnar epithelium forming simple tubular glands and deeper racemose glands, identical to the epithelium lining the cardiac portion of the stomach (Fig. 4C). The presence of esophageal mucous glands underneath this columnar epithelium and under the muscularis mucosae is seen in Figure 5C. In 4 of the cases, definite oxyntic cells were found in the prefixed gastric mucosa, and this was confirmed by stain-



FIG. 6. Case IV. Barium meal showing hiatus hernia with a tumor above obstructing the esophagus. The endoscopically determined mucosal transition is indicated by the arrow.

TABLE III
DIAGNOSTIC FEATURES OF 27 CASES OF ALLISON AND JOHNSTONE'S ANOMALY

	Directly Diagnosable	Diagnosable by Inference		Not Diagnosable
Diagnostic Features	Mucosal pattern showing gastric rugosity	Stricture above inferior pulmo- nary vein level or above the cardiac sphincter mechanism	Barrett's ulcer	No rugosity No stricture No Barrett's ulcer
No. of Cases	4	17	7	4

ing the sections in a special way. In 2 of these cases oxyntic cells were found at the level of the left main bronchus, and, therefore, high up in the segment of gullet lined with gastric mucosa, quite near to the transition, which in both of these cases lay at the aortic arch. This is contrary to the findings of Allison and Johnstone in 1953, who said that oxyntic cells were entirely absent in the segment in question. Barrett in 1958 stated that they may occur at the lower end. The 2 cases cited here indicate that they may occur at the upper end. Nevertheless, it is important to stress that the presence of oxyntic cells anywhere in the prefixed gastric mucosa is exceptional.

ROENTGENOLOGIC FEATURES

In reviewing this large number of cases certain facts about reflux esophagitis became crystal clear. Because the ulcer of reflux esophagitis affecting squamous epithelium represents only a superficial desquamation, it can never be seen as a roentgenologic ulcer, and cannot, by its very nature, be represented by a barium rest or ulcer niche. Indeed, the only roentgenologic manifestation of reflux esophagitis is stricture. The lower limit of this stricture marks the mucosal transition.

Without a complicating stricture from reflux esophagitis, the mucosal transition cannot be determined. In none of these cases was there any evidence of sharp change in mucosal pattern, nor was there any contractile ring to mark the spot. The uncomplicated case is, therefore, undiag-

nosable roentgenologically and in some cases the diagnosis can only be made by the endoscopist as a chance finding, which proved to be so in some of the cases in this series. The diagnostic features are summarized in Table III.

Twenty of the 27 cases had an associated hiatus hernia. All of these were of the short esophagus type excepting 2 which were paraesophageal. All cases personally examined had hiatus hernia. All but 2 of the cases with peptic esophagitis had associated reflux. These 2 exceptions would indicate that occasionally the gastric mucosa lining the gullet may produce enough acid pepsin unaided to digest the squamous epithelium above it.

A comparison was carried out of the levels of the mucosal transition of a large number of cases of simple hiatus hernia with cases which had a portion of gullet lined with gastric mucosa. The result of this comparison is shown in Figure 7. It will be seen that in the uncomplicated cases of hiatus hernia, the mucosal transition is never higher than the lower pulmonary vein at 30 cm. In only 3 of the cases in which a portion of gullet was lined with gastric mucosa was the transition as low as the lower pulmonary vein. In all the other cases, the transition was higher. Three of the cases had hardly any squamous epithelium in the gullet at all. It is possible to state clearly that if the mucosal transition is above the lower pulmonary vein level at 30 cm., then a portion of gullet is lined with gastric mucosa.

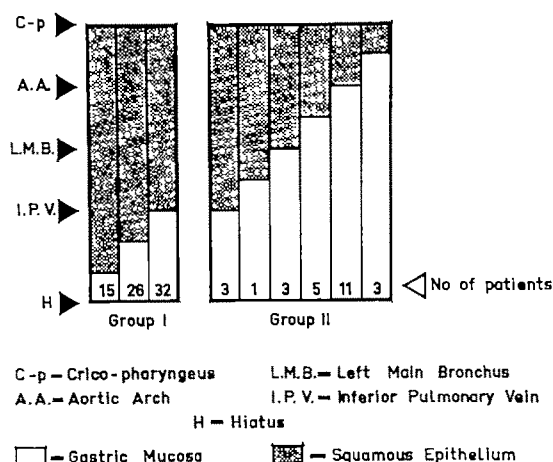


FIG. 7. Comparison of the levels of the mucosal transition of 26 cases of Allison and Johnstone's anomaly, with 73 cases of uncomplicated hiatus hernia. It will be noted that the mucosal transition in Allison and Johnstone's anomaly is never lower than inferior pulmonary vein level, and, if the transition is above this level, then a portion of gullet is lined with gastric mucosa.

BARRETT'S ULCER

There were 7 cases in all, 5 of which had an associated hiatus hernia. In only 1 case was there no gastroesophageal reflux. Stricture formation caused by Barrett's ulceration was present in 2 cases.

The roentgenologic diagnosis of Barrett's ulceration is usually difficult. An ulcer niche or barium rest in the gullet can be simulated by a diverticulum or a pseudo-diverticulum or barium in the rugose folds of gastric mucosa in a hiatus hernia above the diaphragm. In the absence of radiating mucosal folds or stricture formation, the diagnosis can only be made in conjunction with the endoscopic findings, and if the endoscopist finds that the mucosal transition is well above 30 cm., then the chance that the appearance of ulceration is genuine is greatly enhanced. There is always the possibility that a gastric ulcer in the hernia itself may be wrongly described as a Barrett's ulcer.

It is also worth noting that simple ulceration of the esophagus, visible as a barium rest or ulcer niche, never occurs in normal gullet, but only when it is lined with gastric mucosa. Therefore, if a deep penetrating

ulcer is proved in relation to gullet, then that portion of gullet is lined with gastric mucosa. In most cases the diagnosis can only be made when these complications develop. In 23 of the cases, the prefixed gastric mucosa produced a mucosal pattern of fine vertical lines which we associate with squamous epithelium, and it is clear that in the vast majority of cases the condition cannot be diagnosed directly.

ETIOLOGY

Columnar epithelium normally occurs in the gullet in 2 types of glands.

1. *Superficial Cardiac Glands.* These occur at the upper end between the cricoid and the 5th tracheal ring, and in the last few millimeters of the lower end. Although superficial, these glands have a covering of squamous epithelium.

2. *Deep Esophageal Mucous Glands.* These are distributed sparsely and unevenly throughout the esophagus, are 200 to 300 in number and lie in or below the muscularis mucosae (Fig. 5C).

It is of interest to note that if the esophagus be defined as the tubular viscus between the pharynx and the stomach, then the lower 2 cm. is lined with columnar epithelium.¹²

Columnar epithelium can occur abnormally in various parts of the esophagus, and various explanations have been offered to account for this. The first description of ectopic gastric mucosa in the gullet was by Schmidt¹⁰ in 1805. He described islets of gastric mucosa, some of which were large enough to be visible to the naked eye, and nearly all of which were at the upper end of the gullet.

Oxyntic cells may be present, but no definite pathology has ever been associated with these islets, with the possible exception of 1 case of adenocarcinoma described by Carrie⁷ in 1950.

The presence of columnar epithelium in the gullet can be explained on embryologic grounds. This has been studied by Johns¹⁶ in 1952, who says that the entodermal

epithelium of the esophagus is composed at one embryologic stage, of small columnar ciliated cells replaced later by squamous epithelium, and the replacement starts at the middle of the gullet and works out towards either end. Failure of completion of this process would result in columnar epithelium occurring at the upper and lower extremities of the gullet. All the reports of heterotopic mucosa indicate that it occurs at the upper end of the gullet. It has been assumed by Barrett that the gastric mucosa occurring in the lower end of the gullet, and in continuity with the gastric mucosa below the diaphragm, is also of congenital origin, probably because of difficulty in imagining it occurring in any other way.

The congenital origin of this prefixed gastric mucosa is supported by some of the cases in this series. Three of the cases had associated congenital anomalies. One had a right sided aortic arch, another had a hare-lip and cleft palate, and a third had congenital aortic stenosis. If paraesophageal hiatus hernias are considered to be congenital, then it is possible to say that 5 of the cases were thus associated.

There are, however, strong objections to the congenital nature of this condition. First of all, it has never been described as a finding in stillborn infants. It has not been satisfactorily explained why nearly all cases of gullet lined with gastric mucosa are associated with hiatus hernia. The reason given by Barrett is that when the condition is complicated by hiatus hernia, with the production of esophagitis, then the condition is brought to our notice. If it were true that this is a fortuitous combination, then many more cases would be discovered accidentally at endoscopy and postmortem examination than is, in fact, the case. The third objection is that if congenitally displaced gastric mucosa occurs only in minute islets at the upper end, why does it always occur in a continuous sheet at the lower? Hayward's position is to accept this condition as acquired until proved otherwise, and this attitude has much to recommend it.

The acquired theory is based on an analogy with erosions of the uterine cervix, in which squamous epithelium becomes displaced by downgrowths of columnar epithelium, as a result of chronic inflammation. Thus, by extrapolation, peptic esophagitis from acid pepsin reflux results in desquamation of squamous epithelium, which is replaced by upward extension of the gastric mucosa of the stomach, which is, of course, resistant to acid pepsin, and repeated attacks of peptic esophagitis result in a climbing mucosal transition. The possibility of metaplasia or regeneration from mucous glands would also be worthy of consideration.

It was not possible to discover in the long term records of our cases any evidence of a climbing mucosal transition, but in 1960, Goldman and Beckman¹¹ described just such a case. The case in question had roentgenologic and endoscopic evidence of a mucosal transition climbing, during the course of 9 years, from the junction of the middle and lower thirds of the esophagus at 33 cm. to the level of the aortic arch, or above, at 22 cm. The roentgenographic evidence, unfortunately, is so poor that it can be discarded. The crucial points of the case are endoscopic and histologic. In 1950, a biopsy at 33 cm. showed both squamous and columnar epithelium, and was considered to be the transition, and in 1959, there was a stricture and esophagitis at 22 cm., and a biopsy showed columnar epithelium at 24, 30 and 32 cm. The weakness of the case is the fact that these observations were made by two separate observers at separate hospitals, separated by a period of 9 years.

The acquired theory is not supported by all the cases in this series. Eight of the cases had no evidence of esophagitis, either endoscopically or roentgenologically. Some of the cases had no history of heartburn or dysphagia, some of the cases had no reflux, and it is difficult to see how they ever could have suffered from repeated attacks of peptic esophagitis in such a way that over two thirds of the gullet became lined with gastric mucosa. From the evidence pre-

sented here, the Scottish verdict of "not proven" is applicable. An attractive solution would be to postulate two types—one acquired and the other congenital.

SUMMARY AND CONCLUSIONS

Gullet lined with gastric mucosa is a clear cut entity and is distinguishable from hiatus hernia with which it is often associated. Since an eponymous title seems inevitable, the term Allison and Johnstone's anomaly has been applied.

The following conclusions have been reached regarding the roentgenologic diagnosis:

1. The uncomplicated condition is largely undiagnosable roentgenologically.

2. The lower limit of the stricture of peptic esophagitis marks the mucosal transition.

3. If the mucosal transition lies above the level of the inferior pulmonary vein at 30 cm., then a portion of gullet is lined with gastric mucosa.

4. If the stricture of peptic esophagitis is shown to be above the constriction of the cardiac sphincter mechanism, then a portion of gullet is lined with gastric mucosa.

5. Ulcers of gullet manifest as ulcer niche or barium rest are always in gastric mucosa lining that gullet.

The acquired and congenital theories of causations have been discussed.

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STRICTURE OF THE ESOPHAGUS DUE TO NASOGASTRIC INTUBATION*

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INFLAMMATORY diseases constitute the most frequently encountered abnormalities of the esophagus.^{10,11} Most of these lesions subside spontaneously without residual change; an occasional case will proceed to fibrosis or even subsequent stricture formation. The most frequent cause of esophagitis leading to stricture is hiatus hernia with reflux.¹⁰ Among the rarer causes is the subject of this report, namely, esophageal irritation due to repeated or prolonged intubation with a nasogastric tube.

REPORT OF A CASE

R.N., a 31 year old white male, was admitted to the hospital with a 2 month history of rectal bleeding and increasing constipation. Rectal examination and proctoscopy revealed a mass lying 6 cm. above the anus. After biopsy showed this to be adenocarcinoma, a combined abdominal-perineal resection was carried out on July 23, 1962. On the day of surgery, a nasogastric tube was inserted through the patient's nose; this was kept in place for 2 days at which time it was removed. Within a few hours, however, the patient became distended and vomited. A Levin tube was then re-inserted and was left in place for 6 more days. On the seventh postoperative day with the tube in place, the patient developed intermittent retrosternal pain, but, because of continued abdominal distention, the Levin tube was not removed until the ninth postoperative day.

Within 3 or 4 days following removal of the tube, intermittent dysphagia occurred. This persisted for the next 2 months, but was not investigated because of other postoperative complications which arose; namely, ureteral obstruction and a persistent perineal fistula. When the patient's condition stabilized, a

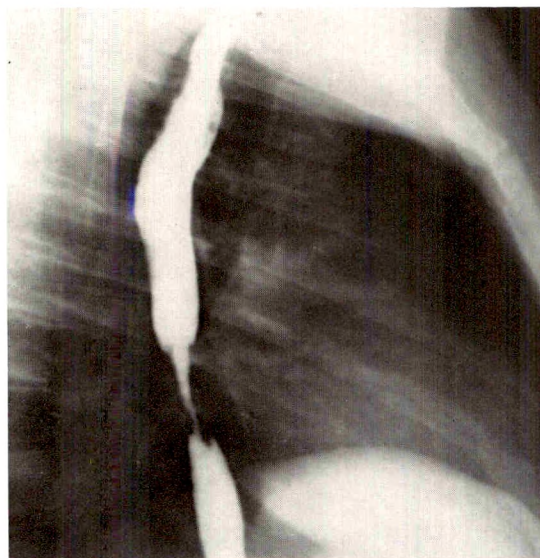


FIG. 1. October 16, 1962: Esophagram done 4 weeks after onset of dysphagia showing annular constricting lesion of mid-esophagus.

barium swallow study was performed; this showed a constricting lesion of the lower esophagus measuring 3 cm. in length (Fig. 1). Esophagoscopy confirmed an annular lesion at this site which was removed through the esophagoscope, leaving slightly denuded but relatively intact mucosa. The specimen consisted grossly of a tubular piece of whitish tissue measuring 3 cm. in length and histologically showed fragments of esophageal mucosa, granulation tissue, and fibropurulent exudate. After esophagoscopy, the dysphagia was much decreased and repeat esophagoscopy on November 15 and December 27 showed only slight narrowing of the esophagus at the site of previous abnormality. The mucosa was completely intact. Barium swallow studies have shown subsequent return of the esophagus to normal with only minimal residual narrowing at the site of the previous stricture (Fig. 2 and 3).

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The views expressed herein are those of the authors and do not necessarily reflect the views of the U. S. Air Force or the Department of Defense.

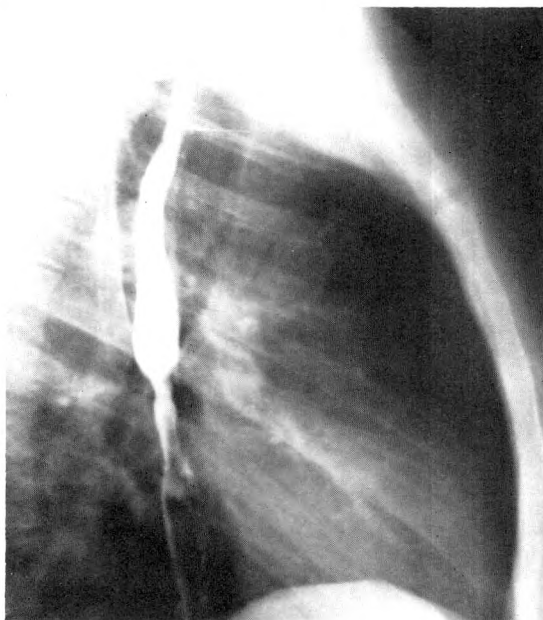


FIG. 2. November 28, 1962: Roentgenogram taken 6 weeks following removal of granulation tissue shows increase in caliber of esophagus.

DISCUSSION

The question of whether esophageal intubation is a true cause of stricture has been a subject of controversy in the literature since Vinson¹¹ advanced the possibility in 1941. From the numerous articles written on this subject, the present authors have collected a unique group of patients, who, without history of esophageal disease, developed esophageal stricture following abdominal surgery and a postoperative course that included a period of nasogastric intubation.

Table 1 summarizes these case reports along with our new case.

Of these patients, almost two-thirds were admitted for duodenal ulcer disease, and all but one underwent a major abdominal surgical procedure. All of the 16 patients were intubated during hospitalization for varying lengths of time. Only 6 patients developed postoperative vomiting, and in only 3 of these was this symptom severe. Four of the group were shown to have a hiatus hernia, but in only 1 was it demonstrated before hospitalization; in the re-

maining 3, preoperative esophagrams were normal.

Since the nasogastric tube was used in all of the patients recorded, we feel that esophageal intubation played a significant role in the production of the stricture.

This opinion has, however, not been shared by all of the previous authors.

Benedict and Gillespie¹ felt that peptic ulcer disease predisposed to esophagitis and subsequent stricture formation because of increased secretion of gastric juices and increased probability of reflux. Although other investigators^{3,12} have reported an increased incidence of esophagitis in patients with peptic ulcer disease, we do not feel that this is of crucial importance since peptic disease was not present in all of the patients summarized.

Douglas⁴ and Larson *et al.*⁵ have suggested that the act of violent vomiting of gastroduodenal juices was an important etiologic factor; however, less than half of the recorded patients showed any vomiting at all, and in only 3 of these was it severe.

Straus⁹ has proposed that an inflam-



FIG. 3. April 18, 1963: Barium swallow study shows return to normal of the esophagus.

TABLE I
PATIENTS WITH ESOPHAGEAL STRICTURE

Authors	Case No.	Age	Sex	Admission Diagnosis	Operative Procedures	Post-operative Vomiting	Nasogastric Intubation (days)	Stricture Site from Incisor Teeth (cm.)	Hiatus Hernia Reflux	Onset of Dysphagia (weeks)
Vinson ¹¹ 1941	1	35	F	Uterine prolapse	Pelvic surgery	0	14	29.0	0	2-3
	2	60	M	Duodenal ulcer	Gastroenterostomy	+++	30	37.0	0	3-4
	3	65	M	Duodenal ulcer	Repair of perforated ulcer	0	Intermittent	37.0	0	8
Larson <i>et al.</i> ⁵ 1942	4	60	M	Duodenal ulcer	Gastroenterostomy	0	3	39.0	0	4
Mason and Ausband ⁶ 1952	5	58	F	Cholecystitis	Cholecystectomy	+	3	30.0	0	4-6
Taylor ¹⁰ 1955	6	64	F	Cholecystitis	Cholecystectomy	+++	4	30.0	0	6
Douglas ⁴ 1956	7	52	M	Duodenal ulcer	Repair of perforated ulcer	0	3	41	0	<1 week
	8	60	M	Duodenal ulcer	Repair of perforated ulcer and gastroenterostomy	+++	14	37	0	40+
	9	61	M	Duodenal ulcer	Repair of perforated ulcer and gastroenterostomy	0	7	39	0	4
	10	66	M	Uremia	None	0	14	35	0	<1 week
	11	53	M	Rectal carcinoma	Abdominal-perineal resection	0	5	33	0	16
Bingham ² 1958	12	41	M	Duodenal ulcer	Repair of perforated ulcer	0	2	lower esophagus	0 then +	4
	13	68	M	Duodenal ulcer	Repair of perforated ulcer and gastroenterostomy	+	2	lower esophagus	0 then +	8+
	14	61	M	Duodenal ulcer	Gastroenterostomy and vagotomy	0	2	lower esophagus	0 then +	2
	15	39	M	Duodenal ulcer	Multiple gastric procedures	0	5	lower esophagus	+	3
Waldman and Berlin 1965	16	32	M	Rectal carcinoma	Abdominal-perineal resection	+	7	23	0	4-6

matory process occurring in the upper abdomen combined with surgical trauma produced an ascending retrograde thrombophlebitis with resultant fibrosis and stricture of the periesophageal tissue. This particular view is thought to be quite unlikely.^{4,7}

Bingham² decided that hiatus hernia was the responsible mechanism, but in only 4 of our cases was a hernia present.

The pathogenesis of the production of stricture due to intubation has been attributed to the irritation of passage, or to position, of the tube over a long period of time,^{4,10} or as a result of straightening of the esophagogastric angle allowing gastric juices to trickle about the tube into the distal esophagus.^{4,7} This latter hypothesis has been challenged by the work of Nagler *et al.*⁸ who failed to demonstrate any reflux around nasogastric tubes experimentally in 20 patients. We feel that the most likely etiology of stricture in the cases summarized is direct trauma of passage or prolonged use of a nasogastric tube.

In summary, several etiologic possibilities have been advanced in 16 cases of esophagitis with subsequent stricture formation. Of these, only nasogastric intubation occurred in every case, and therefore, appears to be the most likely cause.

DIFFERENTIAL DIAGNOSIS

The constricting lesion of the esophagus demonstrated in our patient (Fig. 1) closely resembles carcinoma. Indeed, on a purely roentgenographic basis, it would be difficult to differentiate a malignancy from a benign stricture in this case. If this roentgenographic appearance is found in a patient with the history and clinical events described above, the possibility of nasogastric stricture can be strongly suspected. Other causes of benign stricture to be considered are hiatus hernia with reflux, ingestion of caustics, perforation by foreign bod-

ies or instruments, or rarely such diseases as tuberculosis, sarcoidosis or syphilis.

CONCLUSION

A case of stricture of the esophagus following repeated and prolonged nasogastric intubation has been presented. The role of the nasogastric tube in the pathogenesis of stricture is discussed.

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GASTROESOPHAGEAL REFLUX ELICITED WHILE DRINKING WATER—(WATER SIPHONAGE TEST)

ITS CLINICAL CORRELATION WITH PYROSIS

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GASTROESOPHAGEAL reflux is generally recognized as the preponderant factor in the causation of most cases of esophagitis and the accompanying symptom of pyrosis.^{2,9,11} The presence of spontaneous or free gastroesophageal reflux is infrequently demonstrated during the course of a routine upper gastrointestinal roentgenologic study. This is often true even when a hiatal hernia is present.^{9,10,12}

In the majority of individuals with esophagitis, where one might expect reflux, it is not demonstrable during the conventional roentgenologic study. Because the physician has not received expected confirmatory findings of demonstrable reflux from roentgenologists in these instances, he has developed and uses other procedures that are helpful in the establishment of such a diagnosis. These procedures are: (1) esophagoscopy,⁶ (2) the acid perfusion test of Bernstein *et al.*⁴ in which there is infusion of hydrochloric acid into the esophagus with production of symptoms, (3) the aspiration test,⁸ and (4) simultaneous measurement of pH and intraluminal pressures.¹¹ None of these is an especially simple test nor are they all readily available for use by the average practicing physician.

Therefore, any simple standardized roentgenologic technique that could establish the presence of gastroesophageal reflux would be of great value. Such a roentgenologic procedure that can be performed quickly and easily was introduced by de-Carvalho,⁷ a Portuguese surgeon, in 1951. At the completion of the conventional upper gastrointestinal examination, he placed the patient in a supine position and then rolled him to the right about 45 degrees

(Fig. 1). In this position, with the patient's esophagogastric region centered under the fluoroscopic screen, he had the patient drink 100 to 200 cc. of water. The water first clears all residual barium from the esophagus, and then, if reflux is present, gastric barium erupts into the esophagus and the test is considered positive. De-Carvalho refers to this as a "water siphonage" test. Since "siphonage" implies that fluid is being transferred from a lower level over an intermediate elevation, the author's use of the term is inaccurate. More precisely, one might refer to this phenomenon as gastroesophageal reflux elicited while drinking water in the supine position. The author also points out that such reflux does not necessarily occur in all hiatal hernias, and significantly adds that it occurs very often in patients without hiatal herniation of the stomach but in those with the pyrosis syndrome.

A test for eliciting gastroesophageal reflux must have a significant clinical correlation if it is to be accepted as part of a routine gastrointestinal study. To determine whether a correlation with pyrosis existed, 1,000 consecutive upper gastrointestinal studies in ambulatory patients seen in an office roentgenologic practice were analyzed. Age, sex, presence of pyrosis, demonstration of reflux triggered by the patient's drinking water while supine, existence of hiatal hernia and the presence of peptic ulcer, either gastric or duodenal, were tabulated.

METHOD OF EXAMINATION

Following the usual upper gastrointestinal study and immediately after all roent-

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FIG. 1. Patient is supine and turned to the right about 45° . The container with water may be held with the right hand, and the left arm is behind the head and out of the screening field.

genography has been completed, the patient is placed on his left side so that the barium accumulates in the fundus. Considerable barium should have remained in the stomach, but if that organ has emptied rapidly, additional barium should be given. The patient is asked to roll onto his back and then about 45 degrees toward the right side at which time he is given a container with about 200 cc. of cold water in which there is a rather large bore plastic tube from which he drinks when directed to do so. The patient's left arm is moved upwards behind the head and out of the screening field. The right arm is positioned so that he is able to hold the container with the right hand (Fig. 1). The fluoroscopic screen is centered over the gastroesophageal segment. The patient is then directed to drink rapidly as much water as possible. The Trendelenburg position is not used. The water clears all residual barium from the esophagus, and the test is negative if no barium returns to the esophagus from the stomach. The presence of a few trickles of barium-stained water does not constitute a positive test and moreover is rather frequent.

If the test is positive, there is, after a momentary delay, a brief peaking or tenting of the barium filled fundus of the stom-

ach and immediately thereafter, a dramatic filling of the lower esophagus, often to the level of the aortic arch (Fig. 2, *A* and *B*). The quantity of barium that rushes cephalad is variable but often it is massive (Fig. 3, *A* and *B*). Thereafter, it is rather easy to elicit reflux or even to demonstrate a latent sliding hernia although the patient is not drinking (Fig. 4, *A* and *B*). Sometimes, rolling the patient slightly backwards and forwards aids in starting the regurgitant flow. Deep respiratory movements also may be of help. Rarely does the patient notice symptoms at the time of reflux.

In order to correlate the positive and negative findings with pyrosis, each patient examined was queried concerning the presence of heartburn. Pyrosis or heartburn is defined as pain, discomfort, or a burning sensation high in the epigastrium or in the retrosternal area often made worse while lying down, bending, or stooping. When the patient denied the presence of pyrosis on routine questioning, repeated leading questions often evoked a history of this condition at some previous time.

RESULTS

The sex distribution of the 1,000 cases was essentially equal (Table 1). There was likewise about equal distribution between

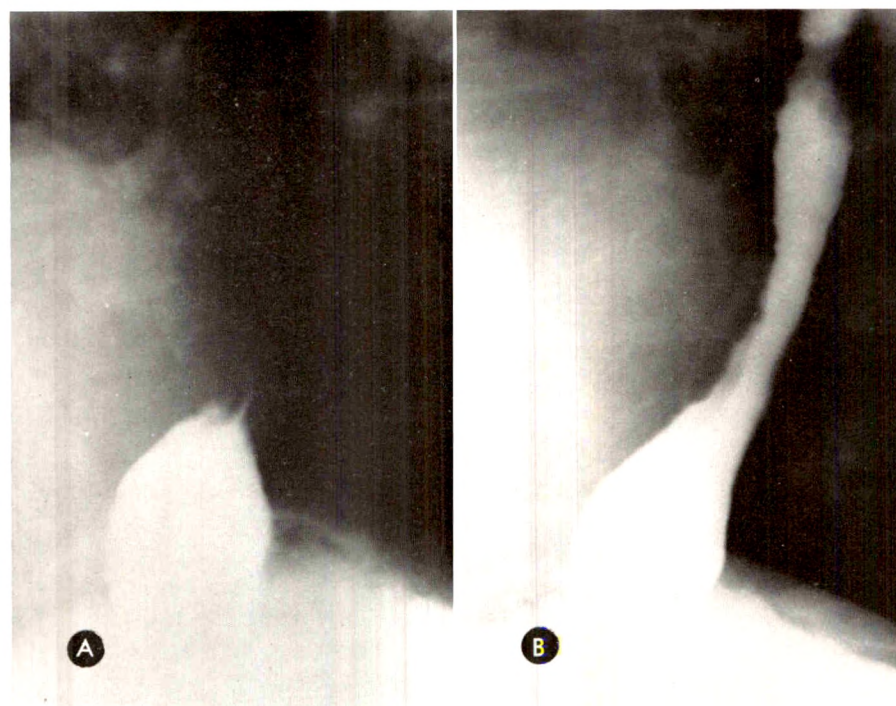


FIG. 2. (A) Reflux begins with barium peaking from the fundus into the lower esophagus. (B) Further reflux carries barium in the esophagus to the level of the aortic arch.

males and females who did show reflux (Table II). The fact that patients between 11 and 20 years of age showed gastroesophageal reflux is not disturbing since these may in later life be those who develop esophagitis and pyrosis. The greatest incidence of induced reflux was between 41 and 71 years of age.

It is noted that 40.5 per cent of all patients exhibited gastroesophageal reflux while drinking water in the supine position and that 57.5 per cent of these had pyrosis; 59.5 per cent did not show reflux, and of this group, only 13.9 per cent had pyrosis (Table III). Therefore, pyrosis was 4.1 times as common in those with reflux.

Of the entire series, 31.6 per cent had some degree of pyrosis, and 73.7 per cent of these refluxed, whereas 68.4 per cent had no pyrosis, and of these only 25.1 per cent refluxed (Table IV). Consequently, 2.9 times as many patients with pyrosis showed reflux than those who had none.

Of the 9 per cent in this series who had hiatal hernias, 78.9 per cent showed roentgenologically demonstrable reflux (Table

v). This is only slightly higher than the 73.7 per cent who had pyrosis but not necessarily an accompanying hiatal hernia (Table IV). Not all patients with hiatal hernias could be made to reflux even with this special procedure.

TABLE I

AGE AND SEX DISTRIBUTION IN 1,000 CONSECUTIVE UPPER GASTROINTESTINAL STUDIES TESTED FOR GASTROESOPHAGEAL REFLUX WHILE DRINKING WATER SUPINE

Age Group	Males	Females	Per Cent of Total
1-10	5	1	0.6
11-20	11	9	2.0
21-30	42	38	8.0
31-40	56	73	12.9
41-50	105	123	22.8
51-60	131	104	23.5
61-70	96	112	20.8
71-80	40	44	8.4
81+	4	6	1.0
Totals	490 (49%)	510 (51%)	100.0

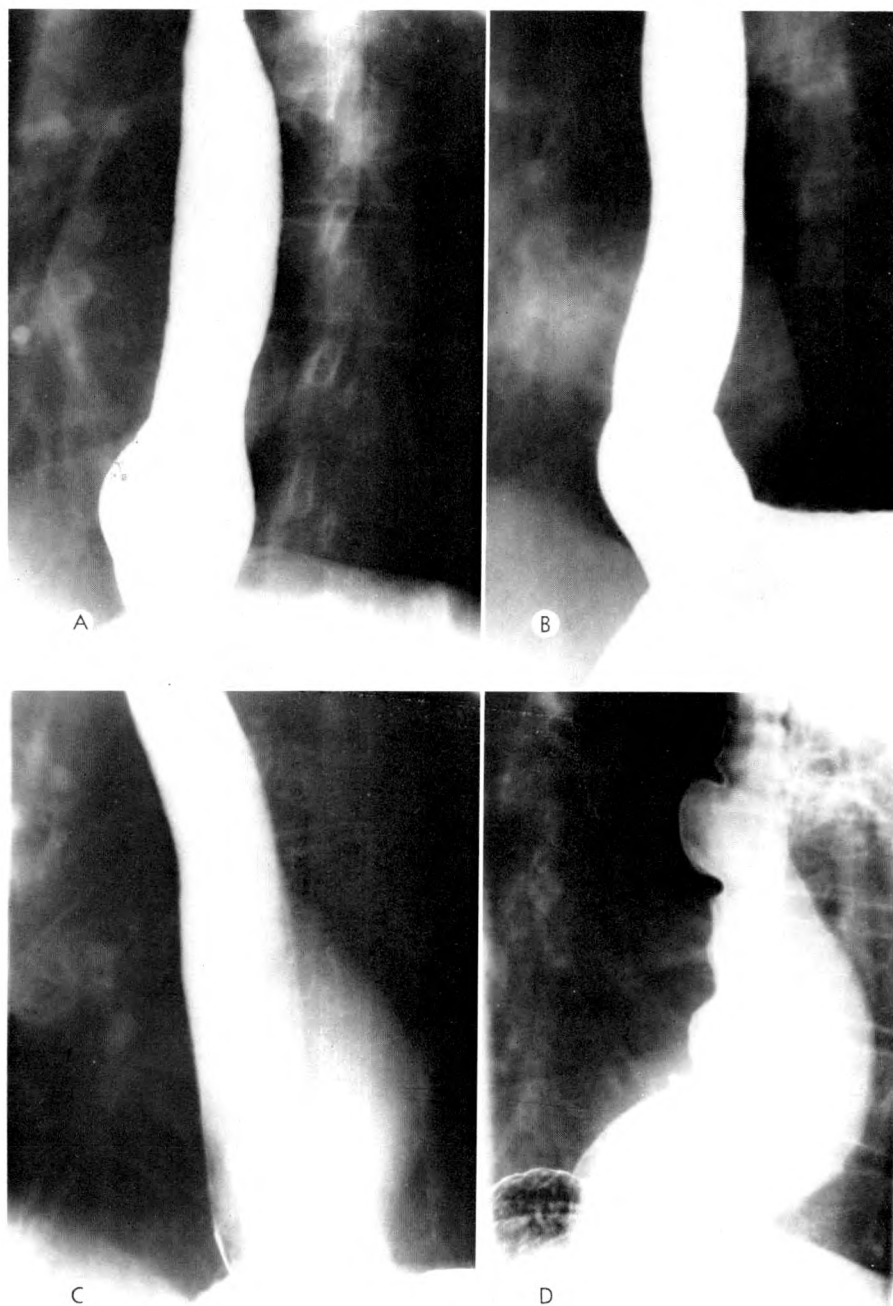


FIG. 3. (*A* and *B*) Gastroesophageal reflux filling the entire infra-aortic portion of the esophagus evoked while the patient was supine and drinking water. (*C* and *D*) Double contrast effect that is imparted in the esophagus in many of these studies.

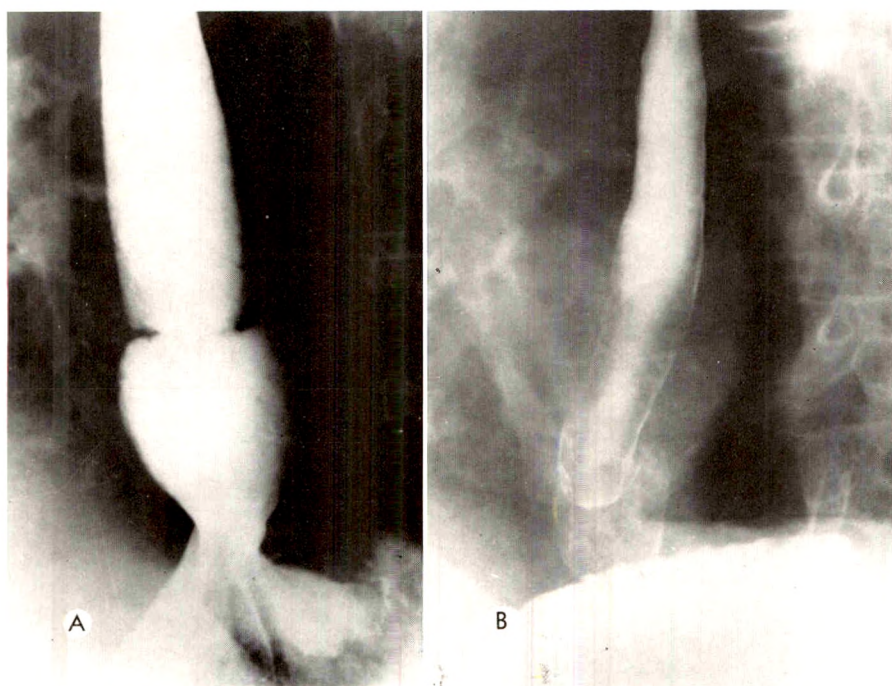


FIG. 4. (*A* and *B*) Sliding hiatal hernias not disclosed until the water technique first showed gastroesophageal reflux.

Eighteen per cent of the patients had peptic ulcer, either gastric or duodenal (Table v). Only 38.3 per cent of these individuals showed gastroesophageal reflux, a figure not significantly different than the 40.5 per cent reflux figure for the entire series (Table III).

Serendipitously, latent sliding hiatal hernias were frequently demonstrated at the time of reflux (Fig. 4, *A* and *B*). Once discovered, it was quite easy to demonstrate these with conventional methods. In instances where small hiatal hernias had been demonstrated prior to the perfor-

TABLE II

AGE AND SEX DISTRIBUTION OF 405 PATIENTS WHO SHOWED REFLUX WHILE DRINKING WATER SUPINE

Age	Males	Females	Total Examined	Total with Reflux	Per Cent of Entire Series (1,000 patients)
1-10	0	0	6	0	0
11-20	4	1	20	5	1.2
21-30	21	11	80	32	7.9
31-40	27	29	129	56	13.3
41-50	44	42	228	86	21.3
51-60	47	33	235	80	19.8
61-70	44	52	208	96	23.7
71-80	24	22	84	46	11.4
81-90	3	1	10	4	1.0
Totals	214 (52.8%)	191 (47.2%)	1,000	405	99.6



FIG. 5. Junctional ulcer is shown with a fully distended esophagus above and herniated stomach below the site of ulceration as gastroesophageal reflux occurred with the patient drinking water while supine and rolled to the right 45°.

mance of the test, these manifest hernias were often more easily seen at the time of reflux.

If reflux occurred while the patient was

TABLE III

CORRELATION OF GASTROESOPHAGEAL REFLUX ELICITED WHILE DRINKING WATER AND PYROSIS (1,000 CONSECUTIVE UPPER GASTROINTESTINAL STUDIES)

	No.	Per Cent	No. with Pyrosis	Per Cent
Patients Showing Reflux	405	40.5	233	57.5
Patients Not Showing Reflux	595	59.5	83	13.9
Totals	1,000	100	316	31.6

TABLE IV

CORRELATION OF THE PRESENCE OF PYROSIS AND GASTROESOPHAGEAL REFLUX ELICITED WHILE DRINKING WATER (1,000 CONSECUTIVE UPPER GASTROINTESTINAL STUDIES)

	No.	Per Cent	No. with Reflux	Per Cent
Pyrosis History or Complaint	316	31.6	233	73.7
No Pyrosis History or Complaint	684	68.4	172	25.1
Totals	1,000	100	405	40.5

drinking water in the supine position, stenotic and ulcerated lesions of the esophagus and the esophagogastric junction could be demonstrated much better than with other techniques because of the complete distention of the esophagus and esophagogastric junction both above and below the lesions by the refluxed, somewhat dilute barium (Fig. 5). The effect here was often that of a double contrast roentgen study since not only was the barium dilute, but there was also sufficient air mixed with it to make the visualized esophagus quite radio-lucent (Fig. 3, C and D; and Fig. 4 B).

COMMENTS

The data presented indicate that the test described for eliciting gastroesophageal reflux correlates quite well with the clinical presence of pyrosis. Brombart⁵ demonstrated a similar degree of correlation.

If this is an acceptable relationship, this relatively simple test can be used in pa-

TABLE V

ASSOCIATION OF HIATAL HERNIA AND PEPTIC ULCER WITH GASTROESOPHAGEAL REFLUX INDUCED WHILE DRINKING WATER (1,000 CONSECUTIVE UPPER GASTROINTESTINAL STUDIES)

	No.	Per Cent of Total	With Reflux	Per Cent
Hiatal Hernia	90	9.0	71	78.9
Peptic Ulcer	180	18.0	69	38.3

tients with pyrosis who are suspected of having gastroesophageal reflux. True, 13.9 per cent of the individuals in this series had pyrosis without showing reflux (Table III). The reason for this is not apparent. Conversely, 25.1 per cent of the patients without pyrosis (Table IV) did show reflux. The explanation for this is conjectural, but several factors should be considered. First, the quantity and the quality (pH) of the refluxed gastric contents would seem important. Second, the frequency and duration⁸ of the reflux are prominent factors. Ardran and Kemp¹ point out that duration of reflux is governed by the normal expirator mechanism in the esophagus. They state that regurgitated material may be immediately expressed by esophageal contractor waves so that gastric contents do not remain in the esophagus long enough to cause esophagitis. Third, the variability of the patient's registration of symptoms must surely be evaluated. Finally, there is the presence of mucus secreted in the esophagus plus the alkaline swallowed saliva, both variable, and both providing some protection against regurgitant acid pepsin.³

The technique of the test is simple and not at all disturbing to the patient. It takes only a minute or two at the completion of a routine upper gastrointestinal study. Since it appears, from the data presented, that reflux is quite reliably correlated with pyrosis, the test might easily be made part of a routine upper gastrointestinal examination in an effort to explain pyrosis and to detect latent hiatal hernias not otherwise demonstrable, as noted above.

SUMMARY AND CONCLUSIONS

Gastroesophageal reflux which occurred with the patient drinking water in the supine position and rolled to the right 45 degrees correlated quite well with the symptom pyrosis. This simple roentgenologic procedure was quickly performed, without objection from the patient, in 1,000 consecutive nonselected upper gastrointestinal studies from an ambulatory office practice.

Of the 40.5 per cent of the patients who exhibited gastroesophageal reflux, 4.1 times as many had the symptom of pyrosis as those who did not have this symptom. Of the 31.6 per cent who had pyrosis, 2.9 times as many patients refluxed as those who did not have the symptom.

With this special procedure, not all patients with hiatal hernias could be made to reflux. Actually, the frequency of reflux in patients with hiatal hernias was not significantly greater than in those with pyrosis alone. When gastroesophageal reflux was elicited with this technique, latent hiatal hernias were frequently demonstrated.

Peptic ulcer patients did not reflux with any greater incidence than those without peptic ulcer.

There was excellent roentgenologic demonstration of stenotic and ulcerating esophageal and esophagogastric lesions when reflux occurred with this test because of the complete distention of the lumen above and below the lesion.

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A ROENTGENOLOGIC STUDY IN MEDIASTINAL ANATOMY AFFORDED BY AIR IN THE MID-ESOPHAGUS

A NORMAL FINDING BUT A POTENTIAL SOURCE OF DIAGNOSTIC ERROR

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THE use of higher voltages and fine-line grids of superior quality for roentgenography of the chest contributes to the visualization of additional mediastinal structures, all of which need anatomic analysis in order to determine whether they are normal or abnormal (Fig. 1).¹⁻⁴

The passage of air down the esophagus is a normal process, accounting for most of the gas present in the gastrointestinal tract. It is surprising, then, that radiologists have paid so little attention to it, except when it occurs in "abnormal" amounts (aerophagia). We have previously dealt with air

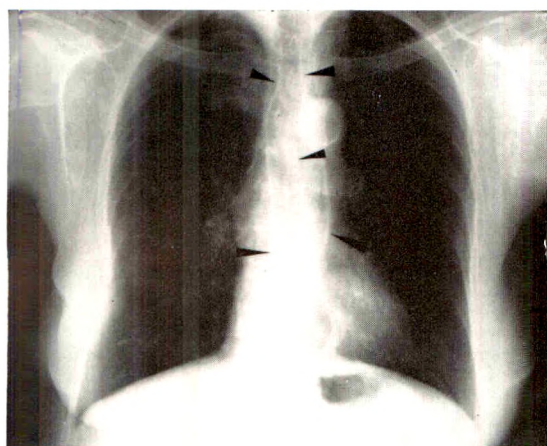


FIG. 1. Entire esophagus distended with large amount of (regurgitant?) gas. Much of the roentgen anatomy is well demonstrated: the pleural-esophageal stripe (upper left arrow) and its continuity with the right wall of lower esophagus (lower left arrow); the posterior mediastinal line (upper right arrow) representing the interface of right and left lung posteriorly; the anterior mediastinal line (middle right arrow); and the left wall of the esophagus, an unusual finding (right lower arrow). (Courtesy of Dr. Arthur L. Thiele, Charlottesville, Va.)

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FIG. 2. Cross section of mediastinum at the level of the carina from cadaver. A represents the aortic arch, B the main-stem bronchi, and E the esophagus, respectively. When the aorta uncoils, it draws the esophagus with it, and, secondarily, the right lung (arrows), thus affording visualization of the proximal lateral wall of the left main-stem bronchus by the air-distended esophagus, and of its medial wall by the right lung. (Modified from Roy-Camille: Coupes Horizontales du Tronc, Masson & Cie, Paris.)

in the esophagus at the thoracic inlet, producing the esophageal-pleural stripe,¹ and with air in the distal esophagus,² a prolongation of the esophageal-pleural stripe. Both of these localizations are frequent and normal findings. The present observations deal with air in the mid-esophagus, a potential source of diagnostic error.

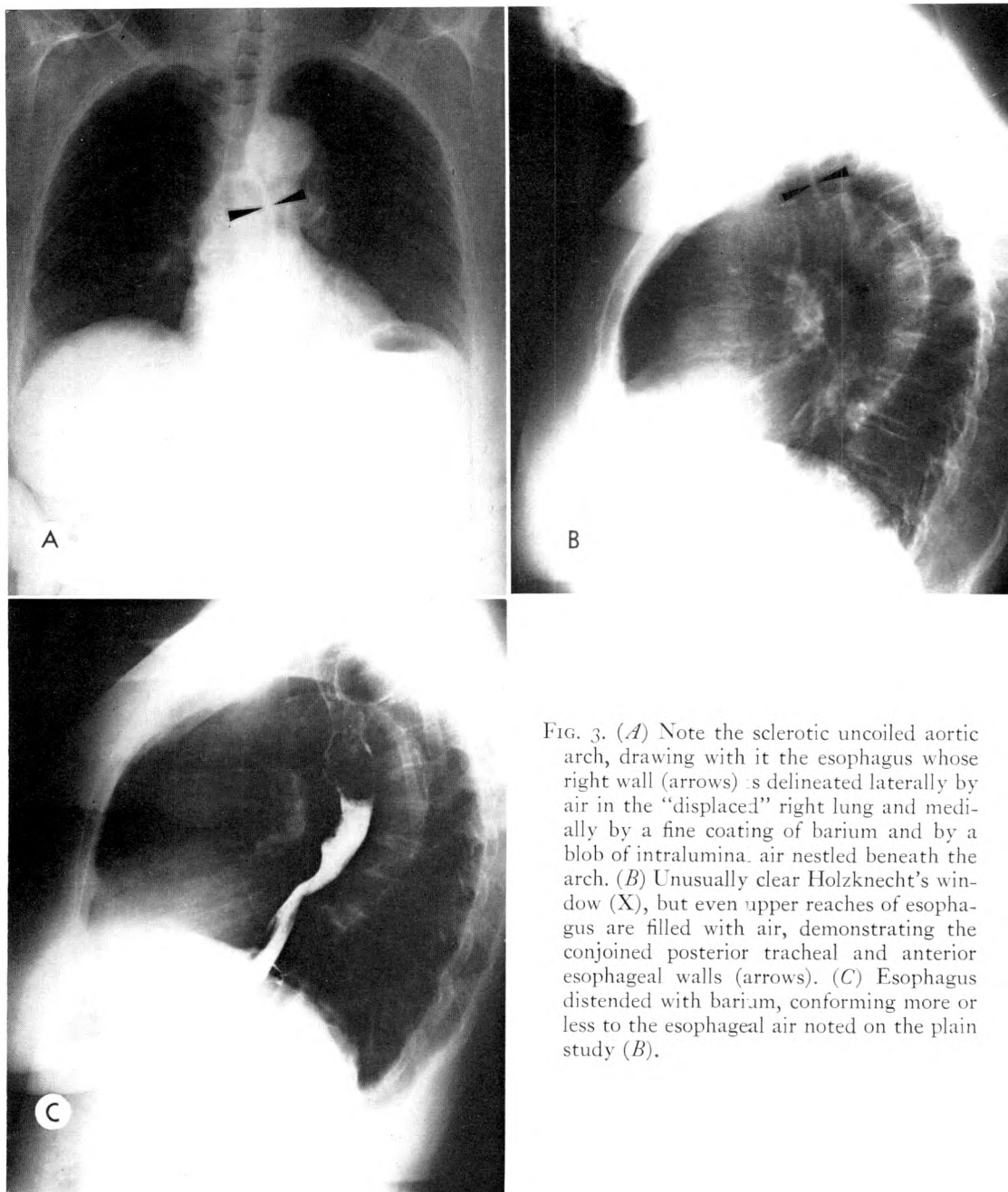


FIG. 3. (A) Note the sclerotic uncoiled aortic arch, drawing with it the esophagus whose right wall (arrows) is delineated laterally by air in the "displaced" right lung and medially by a fine coating of barium and by a blob of intraluminal air nestled beneath the arch. (B) Unusually clear Holzkecht's window (X), but even upper reaches of esophagus are filled with air, demonstrating the conjoined posterior tracheal and anterior esophageal walls (arrows). (C) Esophagus distended with barium, conforming more or less to the esophageal air noted on the plain study (B).

Figure 2 illustrates a transverse section of the mediastinum at the carinal level. It is based on cadaver material, thus not accurately representing the state in life, but adequate for our purposes. Generally, the proximal lateral wall of the right main-stem bronchus is not visualized because of the interposition of the azygos arch between the

air in the right lung and that in the right bronchus; nor is the lateral wall of the left main-stem bronchus visualized because of the interposition of the soft tissue structures of the lung root between the air in the left lung and that in the left bronchus. Neither medial wall is visualized because of the interposed mediastinal soft parts. Any

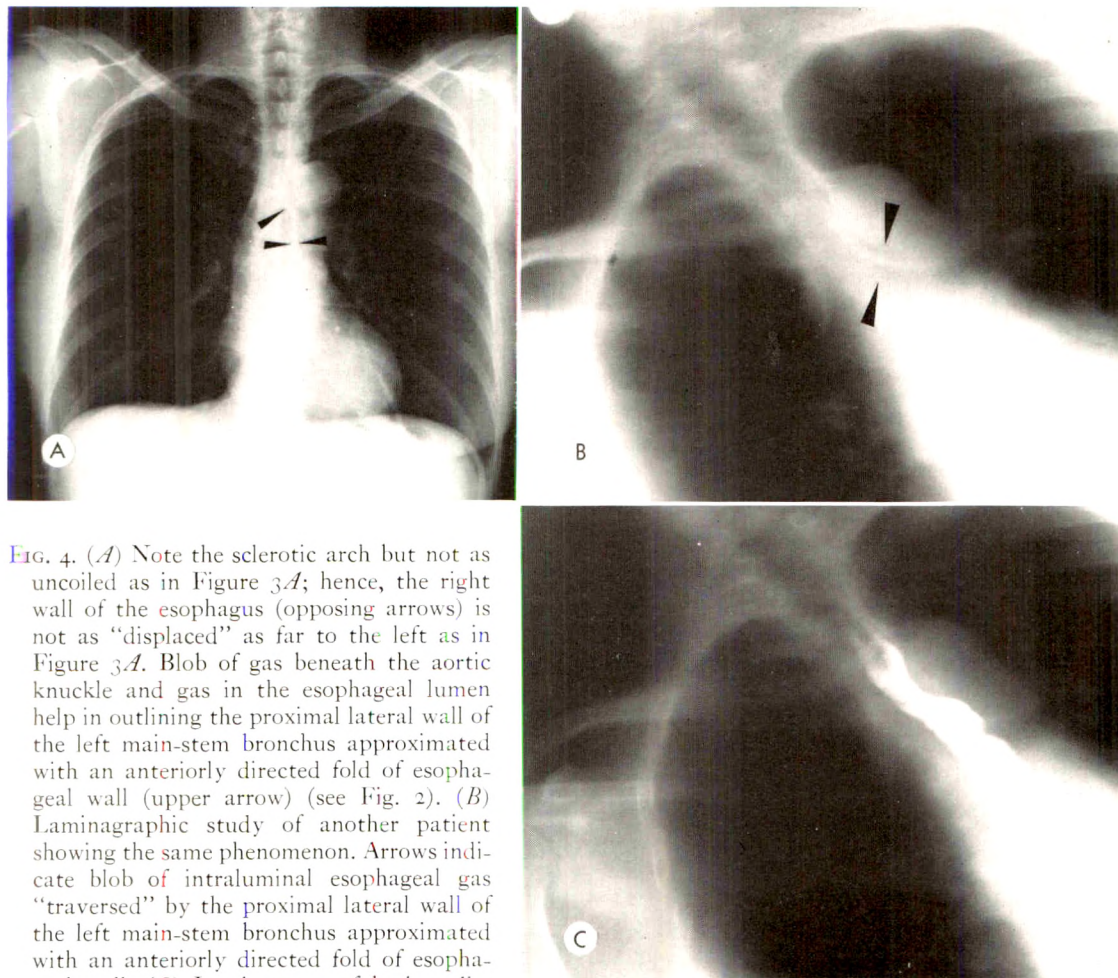


FIG. 4. (A) Note the sclerotic arch but not as uncoiled as in Figure 3A; hence, the right wall of the esophagus (opposing arrows) is not as "displaced" as far to the left as in Figure 3A. Blob of gas beneath the aortic knuckle and gas in the esophageal lumen help in outlining the proximal lateral wall of the left main-stem bronchus approximated with an anteriorly directed fold of esophageal wall (upper arrow) (see Fig. 2). (B) Laminagraphic study of another patient showing the same phenomenon. Arrows indicate blob of intraluminal esophageal gas "traversed" by the proximal lateral wall of the left main-stem bronchus approximated with an anteriorly directed fold of esophageal wall. (C) Laminagram of barium-distended esophagus of same patient as in B. Note the tendency of even barium to be "held up" at this level.

of the bronchial walls may be directly visualized by the calcium within the cartilage.

In older people, the uncoiling aortic arch commonly draws the esophagus with it to the left and posteriorly. Thus, a blob of air within the esophageal lumen may be seen on the roentgenogram of the chest just above or at the level of the carina and to the left, nestled under the aortic arch (Fig. 3A and 4A). The naturally occurring indentation by the left main-stem bronchus upon the distended esophagus, a common finding at barium swallow examination, may play a part in this localization. Exceptionally, the thus displaced esophagus, when distended, can, by its anterior ex-

tension, produce visualization of the lateral wall of the left main-stem bronchus (Fig. 4A). Very exceptionally, the displaced esophagus may permit visualization of the medial wall of the left main-stem bronchus by the air contrast afforded by the simultaneously displaced right lung (Fig. 5). Since the right mediastinal pleura abuts the entire length of the right border of the esophagus, except where the azygos arch intervenes (Fig. 1), simultaneous movement of the right lung with the meandering esophagus can occur.

Roentgenologic detection of gas in the normal esophageal lumen in the lateral view is difficult (except in the cervical esophagus) because there is so little con-

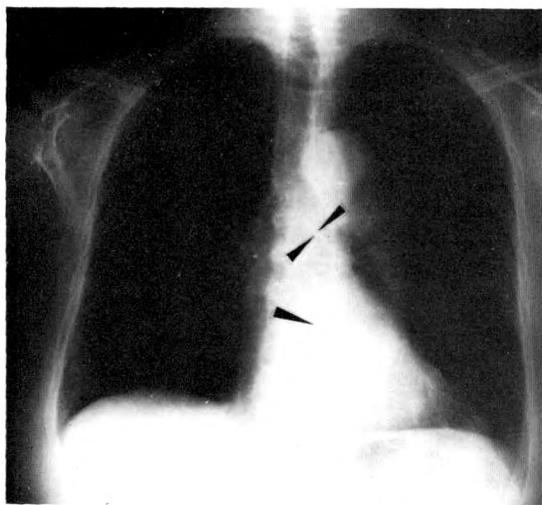


FIG. 5. Single arrow shows the right pleural-esophageal interface, proved as such by a barium swallow examination, "displaced" to the left by the uncoiled aortic arch. Opposing arrows show the medial wall of the left main-stem bronchus, effected by the contrast from the "displaced" right lung and the intrabronchial air (see Fig. 2). This wall is not to be confused with the anterior mediastinal line; the resemblance between the two is only superficial.³

trast with the intrapulmonary air, but such detection is frequently possible (Fig. 3, *B* and *C*). Intraluminal air contributes to the demonstration of an unusually clear Holz-knecht's window (the "clear space" between the concavity of the aortic arch and the heart, anterior and caudal, traversed by the main branch of the left pulmonary artery), best seen on the left anterior oblique or lateral study. Air anteriorly within the trachea and posteriorly within the esophagus allows the direct visualization of the conjoined (by connective tissue) posterior tracheal wall and the anterior

esophageal wall. These two walls may appear distinct roentgenographically when there is a high content of calcium in the tracheal rings or of lipid within the connective tissue. The lipid "line", however, should not be confused with air within the esophageal lumen or wall, or gas dissecting the two structures.

The gas thus visualized in the mid-esophagus and the marked deviation of the right lung to the left in the case of the meandering esophagus may suggest mediastinal hernia, gas-containing lesion, or emphysema, and must be differentiated from these.

SUMMARY AND CONCLUSIONS

Attention to the basic facts of the roentgen anatomy and pathophysiology allows the rational explanation of the visualization of gas nestled beneath the aortic arch and of certain mediastinal structures at the carinal level, especially in older people. The gas, however, must be differentiated from mediastinal hernias, gas-containing lesions, and emphysema.

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BENIGN TUMOR IN GASTRIC AIR BUBBLE*

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RECOGNITION of gastric cancer is an important function of the diagnostic radiologist. It is widely known that polypoid cancer of the fundal area may occasionally be first identified by an abnormal appearance of the gastric air bubble in roentgenograms of the chest.¹ The incidence of recognition in retrospective studies of cancer of the stomach is about 5 per cent.^{2,4} Less appreciated is that benign tumors of the stomach may give evidence of their presence by producing a shadow which bulges into the air bubble in the stomach.

In a review of 82 benign gastric tumors seen at the Ochsner Clinic, we³ encountered a case of benign adenoma of the stomach which was revealed as a soft tissue mass protruding into the medial aspect of the gastric air bubble in a routine roentgenogram of the chest. This case is reported to stress again the importance of carefully studying the entire roentgenogram rather

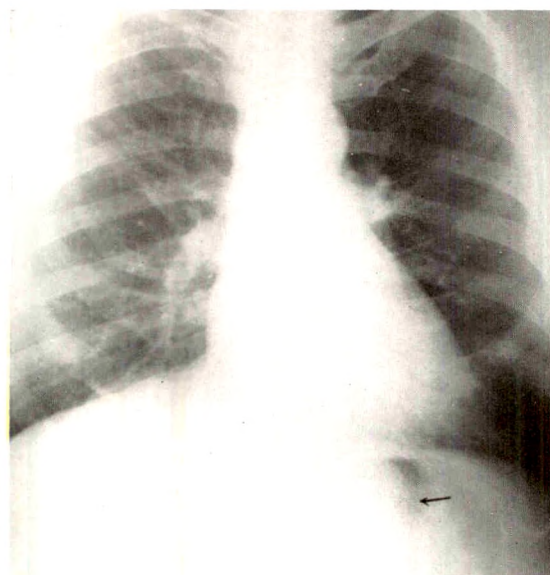


FIG. 1. Roentgenogram of the chest in which a mass protrudes into medial aspect of gastric air bubble.



FIG. 2. Barium study of stomach in which a benign polypoid adenoma is outlined.

than only the lungs or heart. If a lesion is suspected after roentgenography of the chest, gastrointestinal roentgenologic study must be undertaken to confirm it.

REPORT OF A CASE

A man, 74 years old, was examined at the Ochsner Clinic on April 9, 1958 because of gradually worsening shortness of breath and weakness of several weeks' duration. In 1954 he had had transurethral resection for carcinoma of the prostate, and in 1956 he was found to be mildly diabetic. At the time of admission these conditions seemed stable, but hypochromic anemia was present (hemoglobin 6.5 gm. and hematocrit 28 per cent), and occult blood was found in repeated fecal examinations.

On April 19, 1958, no abnormality of the heart or lungs was demonstrable on roentgen-

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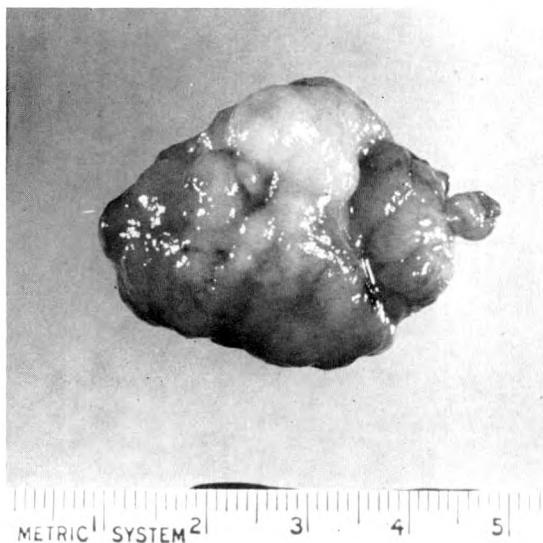


FIG. 3. Photograph of resected tumor.

ography of the chest (Fig. 1), but a soft tissue mass filled the medial side of the gastric air bubble. A gastrointestinal roentgenographic study on the same day confirmed the presence of a mass arising from the posterior aspect of the upper portion of the stomach, near the cardia (Fig. 2).

At gastroscopy on April 28, 1958, a soft mass, thought to be a lipoma was seen in the fundus. At laparotomy on May 8, 1958, a soft, reddish brown, pedunculated polyp was excised. It measured 2.8 by 3 by 1.5 cm. (Fig. 3) and

proved to be a benign gastric adenoma on histologic examination.

On discharge on the ninth postoperative day, the patient was asymptomatic. When last examined 3 years later, his physical condition was good except for diabetes and arteriosclerosis.

SUMMARY

A case of benign polypoid adenoma of the stomach which produced a soft tissue filling defect in the gastric air bubble in a routine roentgenogram of the chest is reported. Contrast study of the stomach confirmed the presence of a tumor, which was removed by local resection. Although common in gastric cancer, this type of roentgenographic visualization seems to be rare in benign gastric tumors. The virtue of conscientious scrutiny of all features on roentgenograms of the chest is affirmed.

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PARTIAL GASTRIC DIVERTICULA*

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THE occurrence of gastric diverticula has been considered to be rare. Nevertheless, with the ever increasing number of roentgenologic studies of the gastrointestinal tract being performed, those found near the gastroesophageal junction have become well known. Diverticula located farther distally are not only very rare but are also less familiar.

By 1951 Palmer²⁴ had collected 412 cases of congenital diverticula from the world literature. In 342 instances, their localization was well documented. Two hundred and fifty-nine or 76 per cent were located in the "usual" position, which was defined as a point on the posterior wall about 2 cm. below the esophagogastric junction and 3 cm. from the neighboring lesser curvature. The others were scattered distally to this point; 15 of these arose from the pylorus and only 9 from the antrum along the greater curvature. A cursory review of the literature since then shows that many more have been reported, among them only 3 in the prepyloric area on the greater curvature.^{23,29,32}

The pathogenesis^{10,12,16,22,28} and classification^{4,24,28,29,31} of diverticula have been adequately covered by others and need no detailed elaboration. Suffice it to state that because of the terminologic definitions, the equation of true pertaining to the congenital lesion and false pertaining to the acquired lesion has not been generally accepted. The terms pulsion and traction diverticula are self-explanatory.

An attempt to correlate specific symptomatology with the diagnosis of gastric diverticula has failed.³⁴ Bleeding,^{5,9,33} perforation,^{20,25} inflammation,^{9,14} and malignant and benign tumors^{1,8,15,19,24} of diverticula have been reported, but, in general, the

elicited symptoms are referable to the gastrointestinal tract. Their discovery is usually incidental to upper gastrointestinal studies or to inspection of a surgical specimen by a pathologist.

The roentgenologic diagnosis is often complicated by the presence of benign or malignant tumors. Aberrant pancreas²⁴ represents one of the most common tissues associated with a diverticulum and is of importance because of possible malignant degeneration,⁶ a feature common to all misplaced structures. Into the same category belong submucosal fibromas²⁴ and adenomas,³⁰ which are associated with congenital diverticula. Carcinomas²¹ and sarcomas² may ulcerate and simulate diverticular pouches; however, the same malignancies^{8,15,18,19} may also arise in the walls of diverticula. The differentiation from benign ulcers may be most difficult at times, especially when diverticula are found distally to the fundus, in areas where the occurrence of other lesions is rather common, while the presence of diverticula is rare. Into this group fall all diverticula in the prepyloric area along the greater curvature, among them the so-called partial diverticulum. This was first described by Samuel²⁹ in 1955 and defined as "... a projection of the mucosa into but not through the muscular coats of the stomach. Inspection of the outer serosal coat of the stomach at operation may show no evidence of any abnormality." Two patients with such diverticula have come to our attention, one of whom was operated upon.

REPORT OF CASES

CASE I. Y.L., 37 years of age, has been treated off and on at the Veterans Administration Hospital in Buffalo, New York since 1960 for

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FIG. 1. Case I. Roentgenogram shows a crater at the greater curvature.

schizophrenic reaction, paranoid in type. In September, 1964 he was re-admitted because of abdominal pain and nausea without vomiting or hematemesis. There was no occult blood in the stools. A gastrointestinal study revealed a crater in the prepyloric area (Fig. 1) at the greater curvature, which was interpreted as an active ulcer. Since there was no improvement



FIG. 2. Case I. Surgical specimen showing the diverticulum.



FIG. 3. Case I. Surgical specimen showing eversion of the diverticulum.

after a period of medical treatment, the patient was operated upon and a subtotal gastric resection was performed. The surgical specimen revealed a partial diverticulum (Fig. 2 and 3). The mucosal pouch protruded into the submucosa but the muscular layer was intact and the serosa was smooth and glistening (Fig. 4).

CASE II. J.H.G., 43 years of age, has had at least 11 admissions to the Veterans Administration Hospital in Buffalo, New York, and was a known alcoholic. He was re-admitted October, 1964 because of vomiting and pain in the epigastrium and in the right upper quadrant. His serum amylase was normal but his urine amylase showed 600 Somogyi units. An upper gastrointestinal study revealed a crater in the



FIG. 4. Case I. Photomicrograph showing a smooth and glistening serosa.



FIG. 5. Case II. Roentgenogram shows a crater similar to that in Case I.

same location and of the same appearance as the one noted in Case I (Fig. 5). A diagnosis of a partial diverticulum was, therefore, made and the patient was not operated upon.

Åkerlund¹ and others^{27,28,30,31} described the roentgenographic characteristics of

diverticula, but these are not all inclusive and apply primarily to diverticula in the neighborhood of the gastroesophageal junction, on which these authors reported. Ulcers and tumors are rather rare in this area and, therefore, a proper diagnosis can be easily secured. Farther distally, the differentiation from an ulcer may be trying. Spot roentgenograms (Fig. 6, A, B and C) showing pliability and varying degrees of distention of the diverticular walls are of utmost importance because an ulcer changes very little in appearance. Small diverticula tend to contract to such an extent that in certain phases of an examination they almost completely disappear (Fig. 6, A, B and C). Mucosal relief studies revealing lack of scarring, swelling, or infiltration and a normal mucosa in the vicinity may establish a proper diagnosis. Whenever pouches do not contract adequately and their walls appear rigid or produce contour defects upon the viscus from which they stem, a tumor must be suspected. Myomata,²⁶ aberrant pancreas, adenomas, neural tumors,²¹ and others may show diverticular pouches of varying sizes which do not always represent ulcers. These pouches may be actually lined with normal mucosa. A differentiation of benign from malignant lesions is, very often, not possible, especially when the tumors are small.

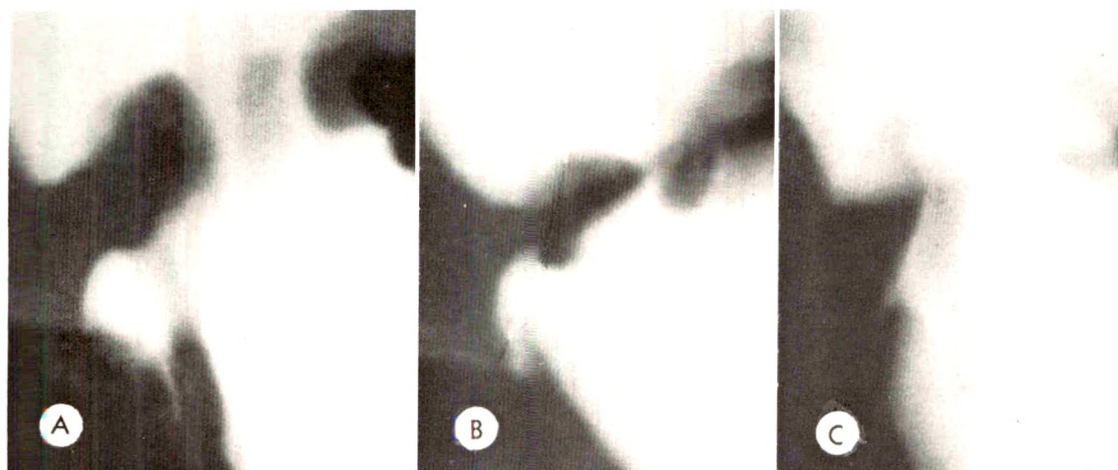


FIG. 6. Case II. (A, B and C) Spot roentgenograms showing pliability and varying degrees of distention of the diverticular walls.

An operative intervention is clearly indicated in these instances, as malignant degeneration is known to occur.

SUMMARY

Prepyloric diverticula along the greater curvature and their diagnostic features are discussed and 2 cases of so-called partial diverticula are reported.

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THE ROENTGENOGRAPHIC SIGNS IN ATROPHIC GASTRITIS AND GASTRIC ATROPHY*

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THIS study was undertaken to evaluate a rather unusual "speckled" appearance of the barium in the fundus of the stomach in some patients as seen in routine roentgenographic studies. We have found that this appearance is frequently associated with hypochlorhydria, atrophic gastritis and gastric atrophy. On the basis of our experience, we feel it is one of the most common lesions in the stomach. In this presentation a correlation of the roentgen signs, the gastric secretory activity and the biopsy findings in 24 patients with atrophic gastritis and gastric atrophy is reported and a new roentgen sign is described.

CLINICAL FINDINGS

A long history of postprandial bloating, vague gas pains, episodic nausea and vomiting—not typical of peptic ulcer diathesis—was given by the majority of the patients. Diarrhea which has long been associated with achlorhydria was not commonly seen.

GASTRIC SECRETORY FUNCTION

Gastric secretory function was determined under conditions of maximum histamine stimulation.⁷ All patients in this series showed marked reduction in the peak secretory rate of hydrochloric acid below the expected 14.5 mEq./30 minute period. This peak secretory rate represents the maximum acid output in milliequivalents in any consecutive 30 minute period following the subcutaneous administration of 50 mg. of Histalog (Fig. 1). Although the majority of our patients would ordinarily be classi-

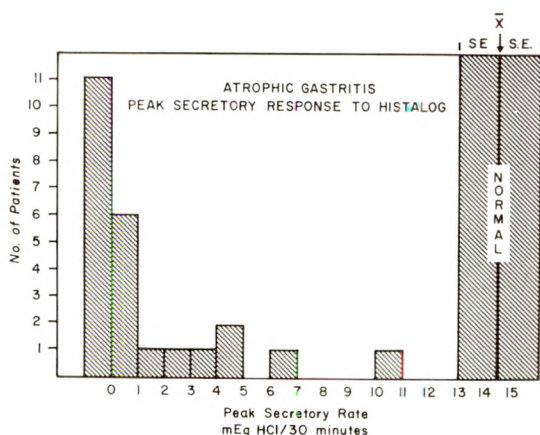


FIG. 1. Atrophic gastritis. Gastric secretory function expressed in terms of peak secretory rate/30 minute period.

fied as having free acid by conventional gastric analysis techniques, a significant reduction in gastric secretory function was present in all cases when subjected to stimulation, equivalent to the "maximal histamine stimulation" (Fig. 2). The peak secretory rate has been shown previously to correlate in a linear fashion with parietal cell mass. For this reason, we have classified gastric atrophy in terms of the percentage of reduction of the parietal cells seen in the mucosal biopsy.

The roentgenographic findings were generally ignored until 1956, when Badenoch *et al.*¹ suggested the correct diagnosis in 20 of 25 cases of proven atrophic gastritis by study of the roentgenograms. Their interpretations were based primarily on the reduction in the size of the rugal folds of the greater curvature of the stomach.

Laws and Pitman,⁶ in 1960, described 4 major roentgenographic signs which they

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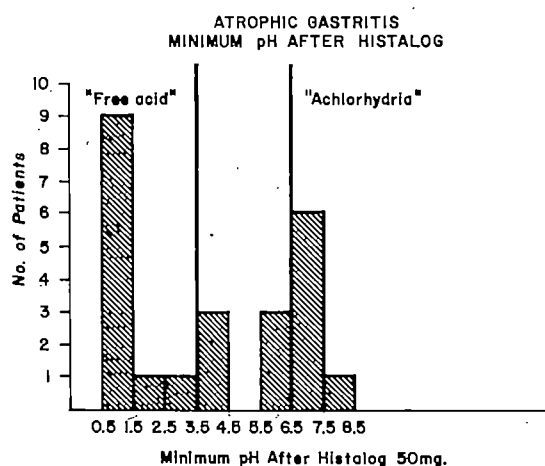


FIG. 2. Atrophic gastritis. Gastric secretory function expressed in terms of minimum pH after Histalog stimulation.

observed in 71 patients with the clinical diagnosis of pernicious anemia. A definite diagnosis of gastric atrophy could be made by a combination of roentgenologic signs in 38 patients (54 per cent) and suggested in 17 others. These signs were: (1) a tubular shape of the stomach in 70 per cent; (2) a smooth greater curvature in 80 per cent; (3) a "bald" fundus in 71 per cent; and (4) an appearance of "tissue paper folds" in the fundus in 31 per cent. A firm diagnosis of gastric atrophy was made if 3 features were seen in any one patient. Age was apparently no factor in the occurrence of these findings. Unfortunately, no biopsies were obtained in this series and varying degrees of atrophy were presumed to be present.⁶

Joske and Vaughan,⁵ in 1962, correlated gastric biopsy specimens with roentgenograms in 160 patients in order to establish

the criteria for the roentgenographic diagnosis of gastric atrophy (radiologic gastric atrophy—RGA). The biopsy specimens were classified into 5 grades on the basis of histologic findings, representing increasingly severe mucosal changes. To the criteria of Laws and Pitman,⁶ and others,¹ they added the additional roentgenographic findings of (1) the hypotonic stomach "H-bomb sign" and (2) the obliteration of the rugal folds when the stomach is distended.

Bock *et al.*,² in 1963, studied the mucosal folds along the greater curvature and noted them to be absent or considerably reduced in number and never more than 0.5 cm. in height. The fundus was smooth or showed small indentations.

ROENTGENOGRAPHIC SIGNS

The relative frequency of the roentgenologic signs in our series was compared with that of Laws and Pitman.⁶ The data were obtained from a study of 24 patients. Three patients with dyspepsia were found to have a lesser degree of atrophic gastritis and no significant roentgenographic findings. The data on the "speckled" fundus and peristaltic activity were additional observations (Table I).

The term "*speckled*" fundus is used by us to describe small disseminated collections of barium, which measure 1 to 3 mm. in diameter, in the fundus and middle-third of the stomach (Fig 3, A and B). This finding is best demonstrated when the fundus contains gas. It occurs with all the barium preparations in common use but is a little better demonstrated with the colloidal

TABLE I

Atrophic Gastritis	Badenoch <i>et al.</i> ¹	Laws and Pitman ⁶	Present Study
Tubular stomach (hypotonia)		70%	71%
Smooth greater curvature	80%	80%	67%
"Bald" fundus		71%	79%
"Tissue paper" folds		31%	41%
"Speckled" fundus			87%
Peristalsis			Diminished, occasionally normal

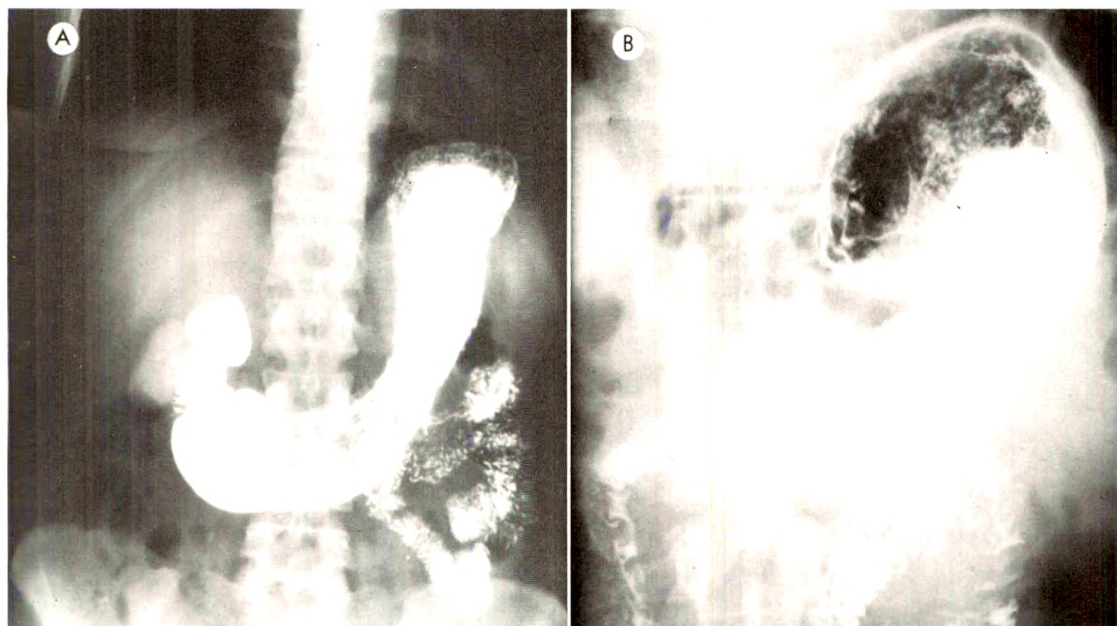


FIG. 3. (A and B) "Speckled" fundus.

type. This was seen to extend into the middle-third of the stomach in several cases, one of which revealed extensive atrophy and small white flecks of secretion throughout upon gastroscopy. It was so marked as to suggest the presence of food or even a bezoar on the initial roentgenographic examination. Repeated studies after gastric lavage and aspiration were relatively unchanged. We think that this appearance is related to the hypochlorhydria and to the changes in the volume and the unusual consistency of the mucus which we have observed in our cases of atrophic gastritis. Studies to determine the exact composition of this material are in progress.

The *tubular stomach* is described by Laws and Pitman⁶ as one with smooth greater and lesser curvatures which are

parallel to one another. This may be demonstrated best in the erect position (Fig. 4). While it may be seen in the normal hypotonic stomach, we think the tubular appearance represents the lack of, or

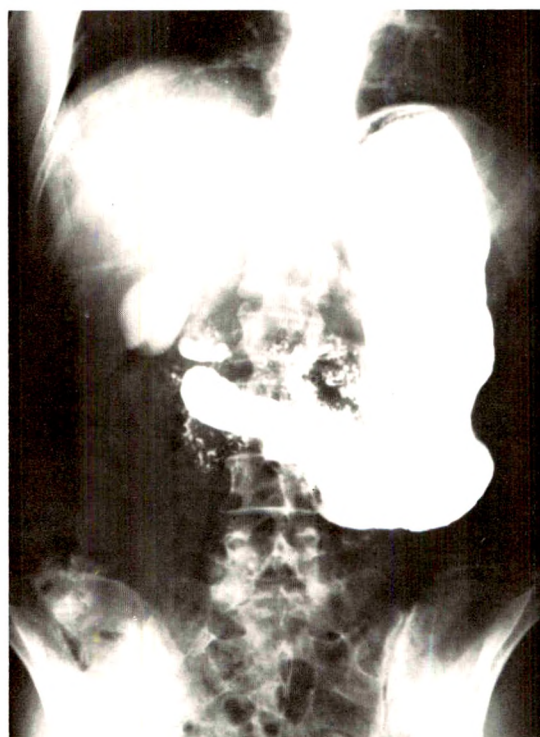


FIG. 4. Tubular stomach—hypotonia, smooth greater curvature.

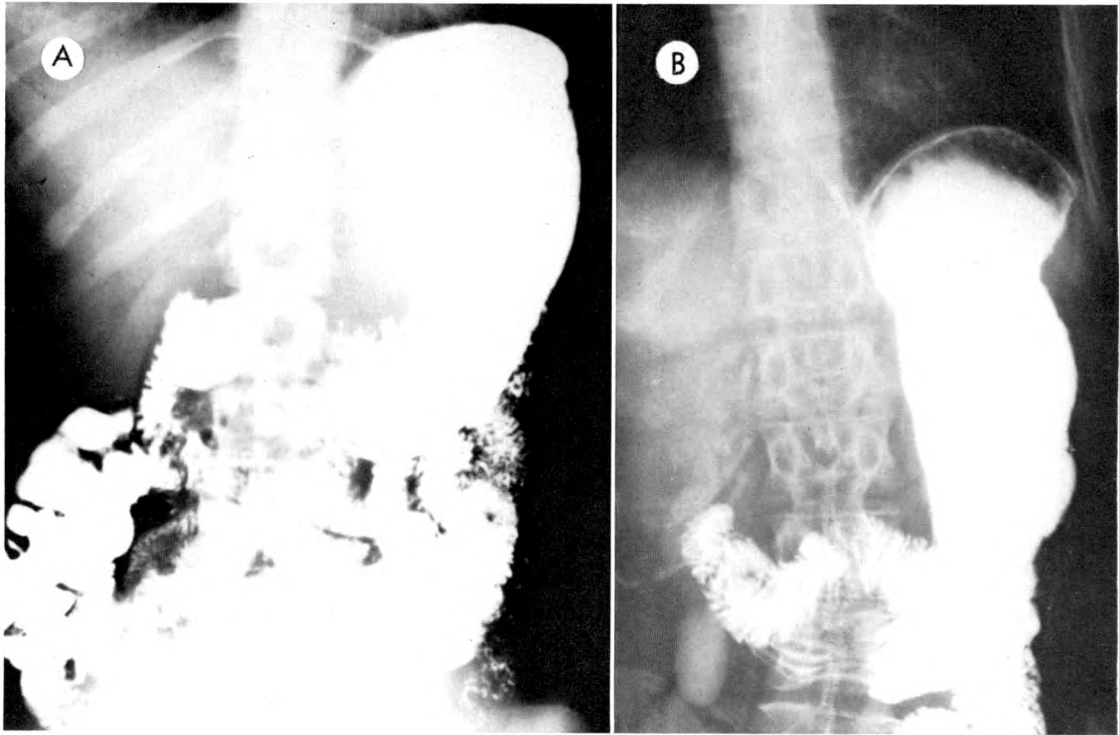


FIG. 5. (A and B) "Bald" fundus.

the reduction in the depth of, the rugal folds along the greater curvature as described by Badenoch *et al.*¹ The patient with the cascade or horizontal type stomach will also demonstrate this tubular appearance but to a less marked degree.

The term "*bald*" fundus is used to describe the absence of rugal folds in the gastric fundus whether it be distended with barium or gas (Fig. 5, A and B). The normal marginal indentations are not present. This finding may be demonstrated in either the erect or recumbent positions and can, occasionally, be recognized at the time of the fluoroscopic examination. Air contrast studies with gas further accentuate the absence of mucosal folds and produce a truly "bald" appearance. The diminished distance from the gas-filled fundus to the superior margin of the diaphragm suggests that the stomach wall is reduced in thickness.

The term "*tissue paper*" folds, as described by Laws and Pitman,⁶ is used to describe thin, crenated mucosal folds re-

sembling crumpled tissue paper (Fig. 6). These are best seen in the fundus and greater curvature when the stomach is almost empty. This was observed in 52 per cent of our cases but was somewhat difficult to evaluate.

Peristalsis was demonstrated to be slightly or moderately diminished in frequency in most cases. Occasionally, it was normal but was not demonstrated to be hyperactive. The capacity of the stomach appeared normal.

Two of the patients in our series demonstrated duodenal bulbar deformity. One was a 61 year old male with a 3 year history of a refractory anemia which responded to androgen therapy. He gave a history of severe peptic ulcer disease 30 years previously, but had no symptoms for several years prior to examination. His weight had remained stable and his appetite was good. The roentgenographic studies revealed the findings of atrophic gastritis and duodenal bulbar deformity (Fig. 7). Mucosal biopsy revealed severe mucosal

atrophy (Grade 3) with chronic inflammation. The peak secretory rate was .26 mEq./30 minutes. Additional studies included serum iron determinations, Schilling tests, blood groupings and gastric cytology, which will be reported elsewhere.

PATHOLOGY

The major observations of these entities have been presented in the English and Australian journals. Joske, Finckh and Wood⁴ reported the histologic findings in a study of 1,000 consecutive successful biopsies obtained from 623 patients by means of a biopsy capsule. Their histologic classification comprised 5 grades, representing increasingly severe mucosal changes. The majority of their patients was being investigated for disease of the gastrointestinal tract, while others had no apparent clinical abnormality. Varying degrees of gastric atrophy were found in about 40 per cent of



FIG. 6. "Tissue paper" folds.



FIG. 7. Duodenal deformity in addition to the demonstration of roentgen signs of atrophic gastritis and gastric atrophy

their biopsy specimens. This finding suggests that these entities are more common than is generally realized.

The biopsy specimens in the present series were classified into 5 grades on the basis of the estimated number of parietal cells which each contained relative to the normal³ (Table II). This classification is arbitrary and the parietal cell estimates are necessarily inexact; however, the specimens available seemed to be divisible fairly accurately into these grades.

TABLE II

CLASSIFICATION OF ATROPHIC GASTRITIS AND GASTRIC ATROPHY BASED ON PARIETAL CELL MASS

Grade 1.	Normal
Grade 2.	Decreased—not to exceed 50% loss
Grade 3.	Decreased—more than 50% but less than 90% loss
Grade 4.	Decreased—loss of 90% or greater
Grade 5.	Total absence of parietal cells

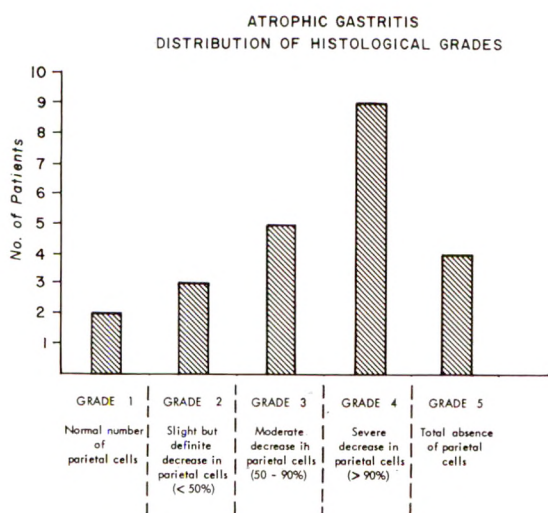


FIG. 8. Atrophic gastritis. Present series. Distribution of cases according to proposed histologic classification.

It cannot be assumed that the findings on biopsy were necessarily representative of the entire gastric mucosa in these cases. Most cases demonstrated the loss of parietal cells to be rather general, but a varied distribution was seen in several specimens.

All atrophic specimens in this series were chronically inflamed. All but one of the specimens in Grade 1 were also mildly inflamed. Mucosal thickness, as measured by microscopic ocular micrometer, varied from 1.0 mm. to 0.56 mm. There was general, but not uniform, correlation between mucosal thickness and the degree of parietal cell loss. Partial intestinalization was a finding in 4 of the severely and completely atrophic specimens. Figure 8 shows the distribution in our series.

Photomicrographs of each grade are

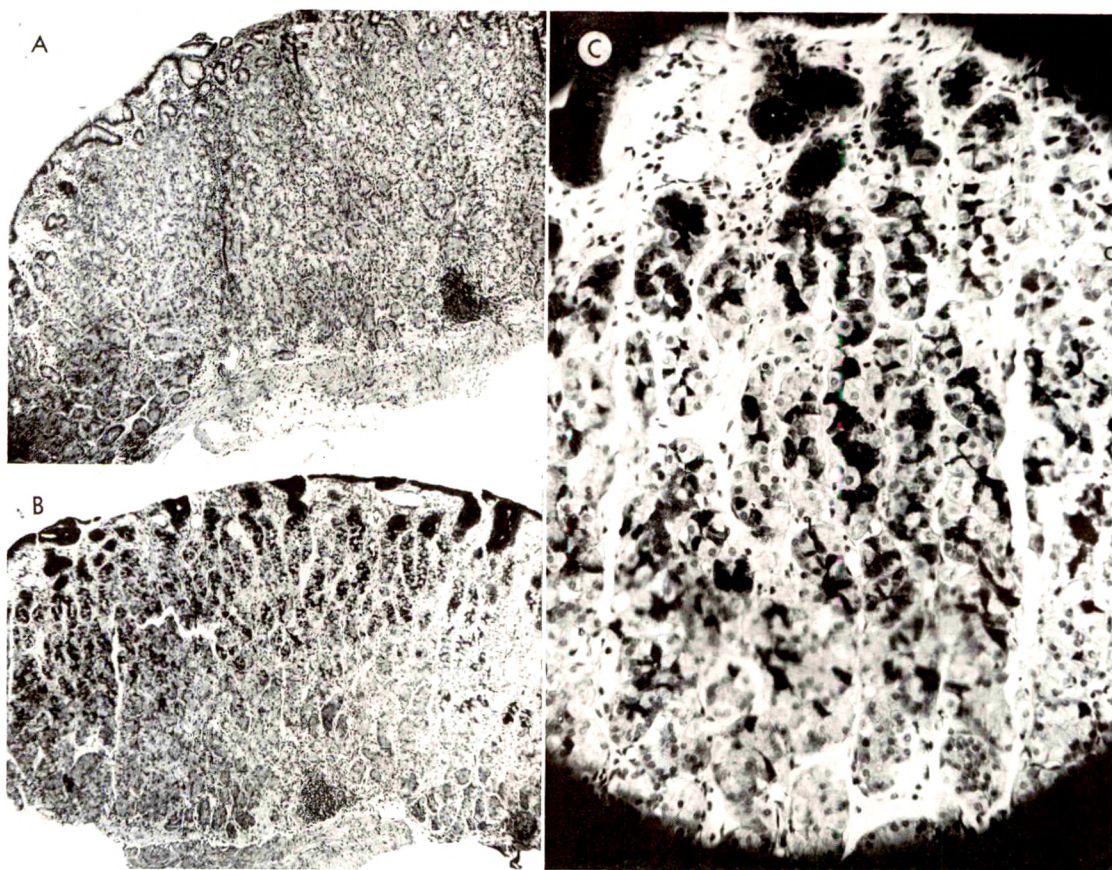


FIG. 9. (A, B and C) Grade 1. Normal. A and B is a magnification of 45 times with a hematoxylin-eosin and PAS stains. C is a magnification of 276 times with PAS stain.

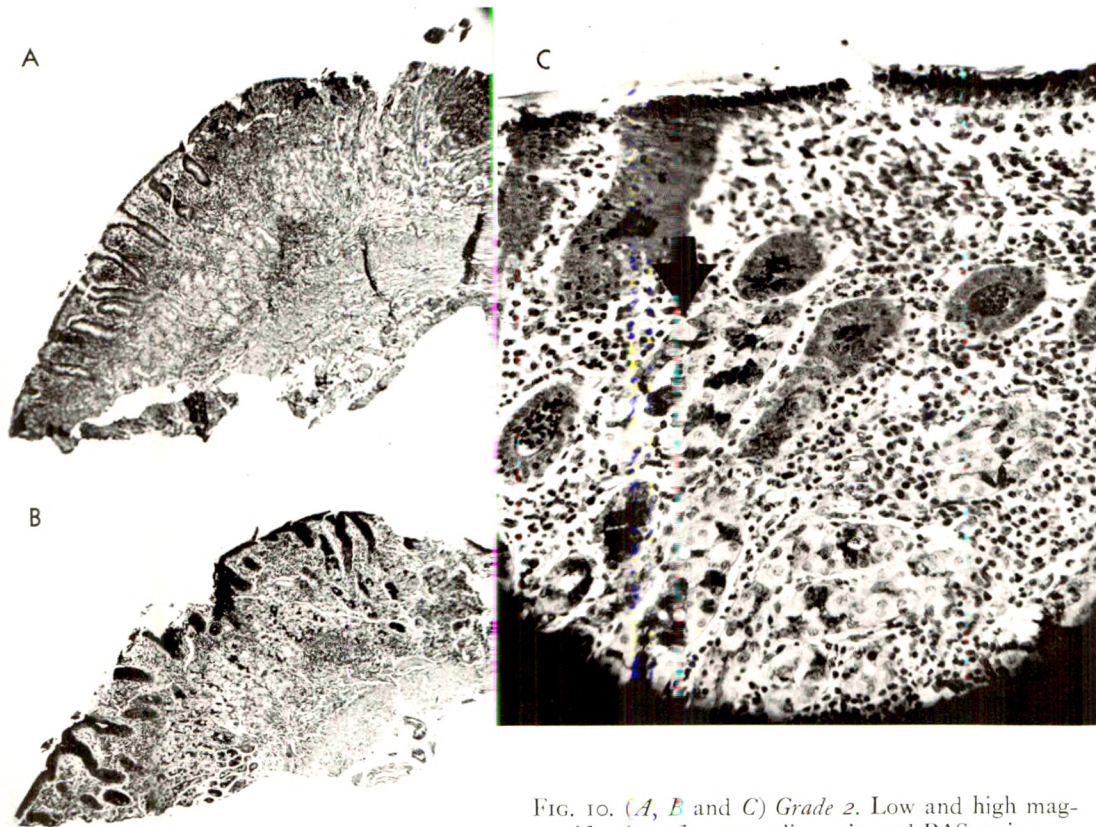


FIG. 10. (A, B and C) Grade 2. Low and high magnifications, hematoxylin-eosin and PAS stains.

presented; some at a magnification of 45 times and others of a more selected area at 276 times magnification.

Grade 1 is an example of the normal gastric mucosa. The partial absence of the surface epithelium is artifactual. The mucosa in the section is 1 mm. thick and has a normal number of parietal cells. The higher magnification shows the necks of the gastric glands in which there are numerous pale-stained parietal cells intermingled with dark red mucous neck cells (Fig. 9, A, B, and C).

Grade 2 shows a definite but slight decrease in parietal cells. The mucosa is 0.75 mm. thick. There is a moderately severe chronic inflammatory process. The superficial defect in the section is artifactual. The higher magnification illustrates a superficial area in the mucosa. Parietal cells

are intermingled with mucous neck cells in the area indicated by the arrow. The prominent chronic inflammatory lymphocytic and plasmocytic exudate is seen in the lamina propria (Fig. 10, A, B and C).

Grade 3 is a typical moderately atrophic specimen. The parietal cells are considerably reduced in number, but are fairly numerous in the mid-mucosa. There is also moderate chronic gastritis with mucosal edema. The mucosa is 0.56 mm. thick. The higher magnification includes the superficial and middle zones of the thin mucosa. The surface epithelium is normal. A small group of residual parietal cells is indicated by the arrow (Fig. 11, A, B and C).

Grade 4 shows a case in which there is almost complete absence of the parietal cells. There is moderate chronic gastritis and the mucosa is 0.66 mm. thick. The

higher magnification reveals only six pale-staining parietal cells. The glands are reduced in number and consist almost entirely of nonspecific mucous cells stained dark red. The chronic inflammatory exudate is evident in the lamina propria (Fig. 12, *A*, *B* and *C*).

Grade 5 shows total absence of parietal cells associated with moderate gastritis and intestinalization. The mucosa is 0.56 mm. thick. The greater magnification shows numerous goblet cells replacing many of the normal epithelial cells. No parietal cells are present. The chronic inflammatory exudate is seen in the lamina propria (Fig. 13, *A*, *B* and *C*).

DISCUSSION

The roentgenographic findings of gastric atrophy and atrophic gastritis in 24 patients are reviewed. The "speckled" fundus which we have described was the most common roentgenographic finding in our experience. Although we are not certain as

to the cause of this appearance, we suggest that it may be the result of changes in quantity or composition of the mucus component of gastric secretion. Further studies of the chemical characteristics of gastric mucus in the patients and its interaction with barium suspensions are needed to provide an understanding of this appearance.

The importance of the diagnosis of atrophic gastritis and gastric atrophy cannot be overemphasized. These patients have a chronic history of gastric symptoms and are constantly seeking an answer and relief of their symptoms. A great deal can be accomplished if a firm diagnosis can be made by gastric biopsy and the patient is reassured that neither gastric ulcer nor carcinoma is present.

Therapy to date, with hydrochloric acid with or without pepsin, has not been adequate. Vitamin B₁₂ and iron have been used without relief. The greatest benefit apparently lies in the fact that a definite

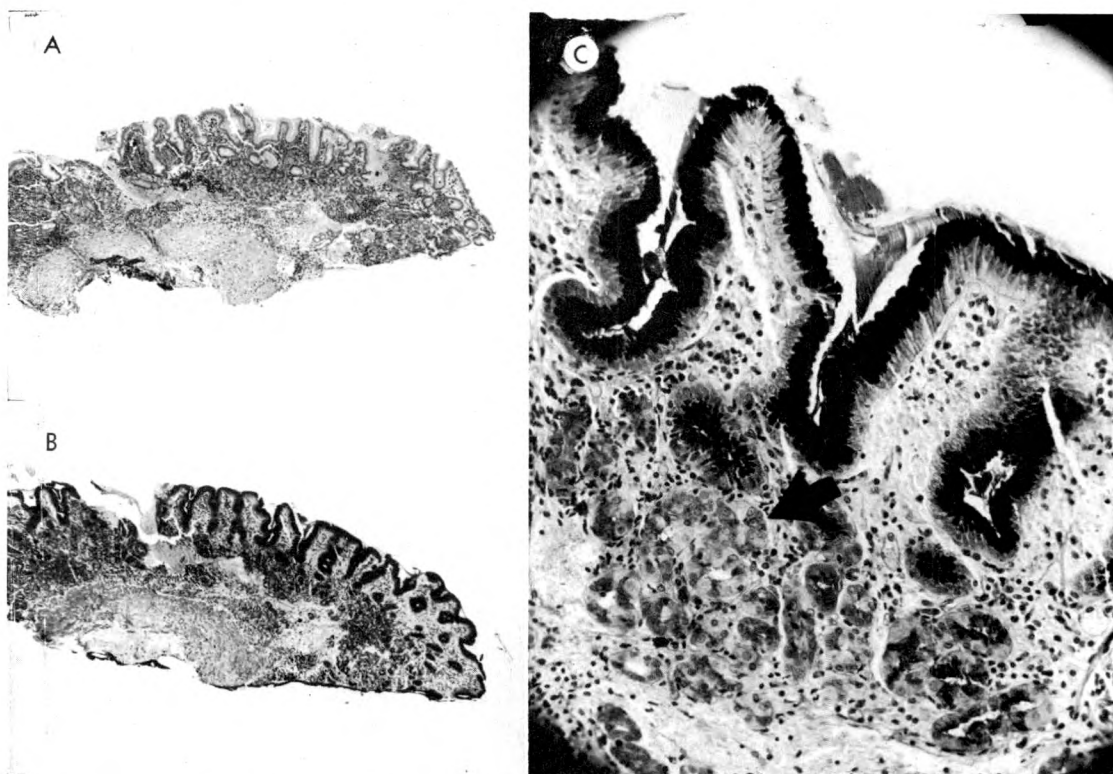


FIG. 11. (*A*, *B* and *C*) *Grade 3*. Low and high magnifications, hematoxylin-eosin and PAS stains.

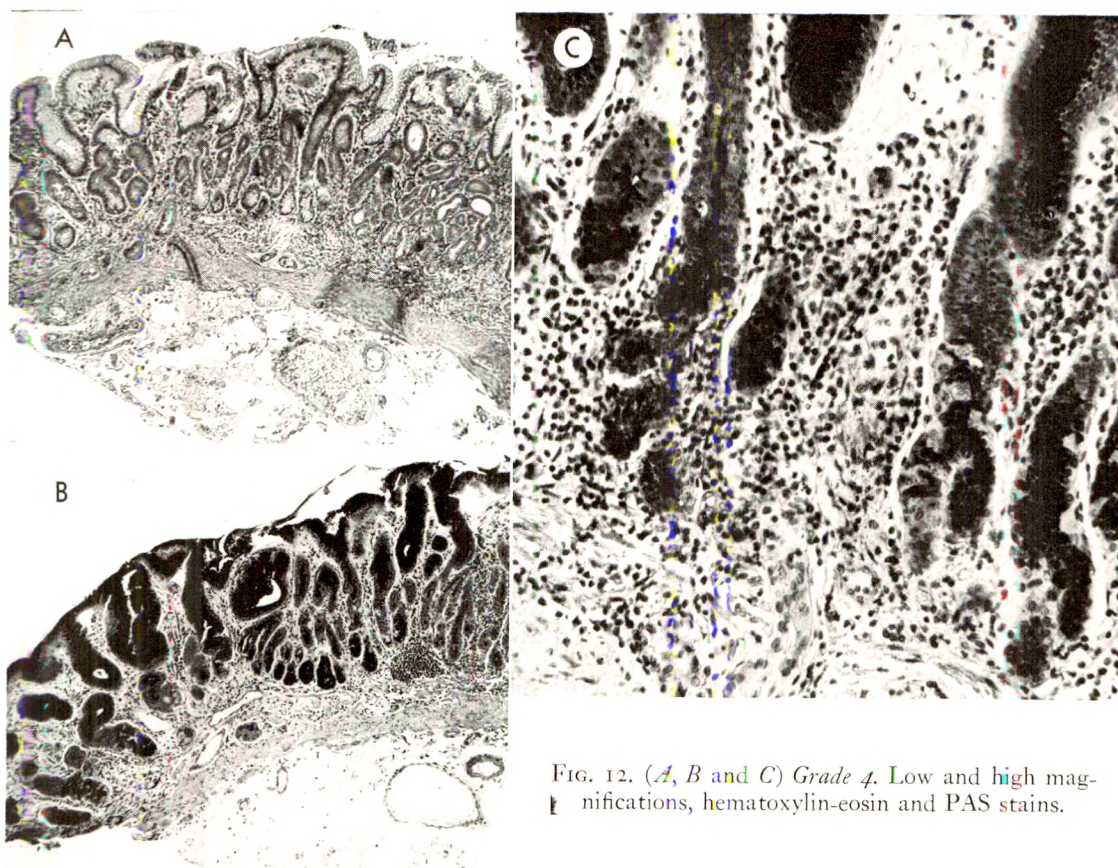


FIG. 12. (A, B and C) Grade 4. Low and high magnifications, hematoxylin-eosin and PAS stains.

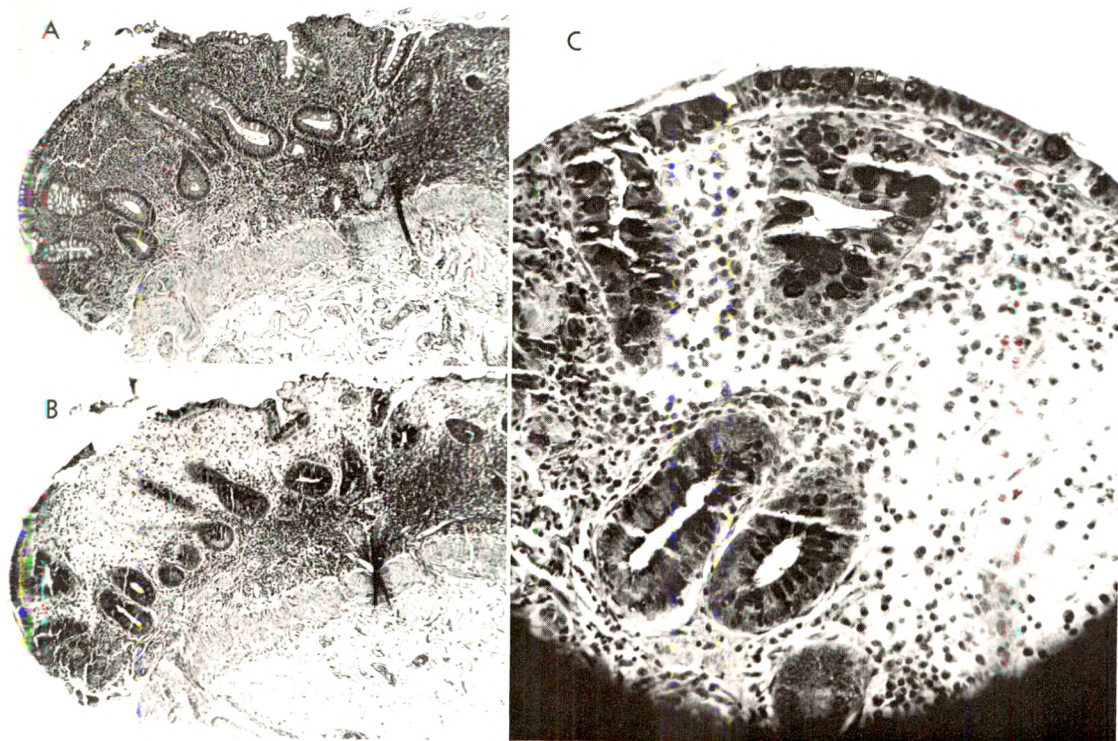


FIG. 13. (A, B and C) Grade 5. Low and high magnifications, hematoxylin-eosin and PAS stains.

diagnosis has been made and the patient knows that there is a basis for the symptoms experienced.

SUMMARY

1. The roentgenographic findings in 24 patients with gastric mucosal atrophy are presented with emphasis on a new roentgen sign, the "speckled fundus."

2. The gastric secretory function in these patients is briefly discussed.

3. A classification of gastric atrophy is given which is based upon the percentage of parietal cells present in the mucosa as seen in fundal biopsy specimens.

4. Fundal biopsy was performed after it was determined that at least 2 of the roentgenographic criteria were present. The correlation appeared to be quite high.

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SITUS INVERSUS OF ALL ORGANS EXCEPT STOMACH*

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ISOLATED inversion of the stomach is a rare anomaly.^{1,2,4} The following case documents a mirror image of this anomaly; namely, inversion of all organs except the stomach. A review of the literature revealed only one other case identical to our patient.⁷

REPORT OF A CASE

A 45 year old white farmer was admitted to the University of Kentucky Medical Center with the chief complaint of low back pain for the past 8 to 10 months with radiation down the left leg. He had known since childhood that his heart was "on the wrong side." Past medical history revealed evidence of hypertension and peptic ulcer. He claimed that his appendix was removed when he was a child. Physical examination showed a blood pressure of 150/90 and a regular pulse of 80. The apex beat of the heart was to the right of the midline. No murmurs, thrills, or arrhythmias were noted. The abdomen showed a right lower quadrant scar. There was tenderness in the epigastric area. There was marked motor weakness of the left lower leg and decrease of the ankle jerk.

A chest roentgenogram showed the cardiac apex pointing to the right (Fig. 1). There were scattered hilar calcifications. The left hemidiaphragm was higher than the right. An upper gastrointestinal series revealed a normal stomach which was posterior to the liver. The liver lobes were inverted. No definite spleen density was seen on the roentgenogram, but a peripheral blood smear demonstrated no changes which would suggest splenic agenesis. The duodenal cap was deformed but no crater could be seen (Fig. 2). A small bowel study showed that the small bowel was within the right half of the abdomen and the cecum in the left lower quadrant. The splenic flexure of the colon was in the right upper quadrant (Fig. 3). A lumbar myelogram showed a complete block at the level of L3. All laboratory studies were normal. The electrocardiogram was consistent with

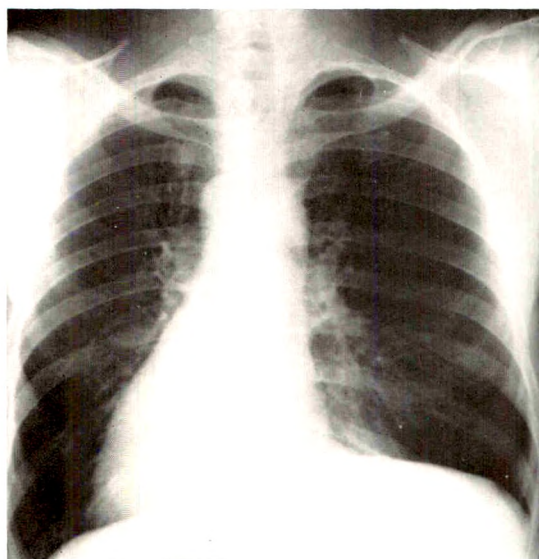


FIG. 1. Chest roentgenogram showing dextrocardia.

dextrocardia. Herniated disks at L4 and L5 were removed, and the patient was discharged 7 days later.

According to the patient, a brother, who died at the age of 3 months, had inversion of one or more organs. His wife and two of his younger children were examined at this hospital and no organ inversion was found. Two older children had had roentgenographic examinations and no anomalies were found.

DISCUSSION

Several cases of isolated inversion of the stomach have been reported.^{1,2,4} In this rare heterotaxy, the right-sided stomach is usually posterior to the liver except where an eventration of the right hemidiaphragm is present, in which instance the stomach is above the liver and may simulate a hydropneumothorax.⁶ Situs inversus of all organs except the stomach, which thus represents a mirror image of isolated inversion of the

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FIG. 2. Upper gastrointestinal roentgenogram showing stomach in normal position but inversion of small intestine, liver lobes and splenic flexure bubble.

stomach, appears to have been reported only once before.⁷

The true incidence of situs inversus in the population is difficult to determine because it is uncommon and its frequency has been studied only in selected groups such as hospital populations and military service inductees. The reported incidence has varied between 1 in 35,000 (0.003 per cent) obtained by LeWald⁵ in studying routine army physicals and 1 in 5,000 (0.02 per cent) in this same author's series as determined at autopsy. The heterotaxy exhibited by the above case must be exceedingly rare since it is an almost unique variation of an uncommon condition.

Since the expression of situs inversus can be total or partial, affecting various organ systems, several different degrees of asymmetric organ inversion have been described. These vary from the reversal of single organs such as in dextrocardia (situs inversus cordis) or isolated gastric inversion to reversal of all body structures such as in situs inversus totalis. Levocardia with situs

inversus, in which the heart is in the normal position with the apex pointing to the left while the abdominal organs are reversed, appears in cases reported to date always to have been associated with complex malformations of the heart of which atrial inversion is common. This is not true for the converse of this heterotaxy; namely, dextrocardia without inversion of the abdominal organs, although many patients with this disposition of internal organs do have heart disease. This difference in frequency of cardiopathy between the above heterotaxies may, however, be more apparent than real since asymptomatic patients with dextrocardia are more likely to undergo barium studies than those with levocardia.

The embryologic and genetic aspects of situs inversus are not well understood. It appears that some forms of situs inversus may be inherited as a recessive trait,³ while others may occur as a developmental anomaly *in utero* and be associated with other congenital defects.

SUMMARY

A case of situs inversus of all organs except the stomach has been reported. This

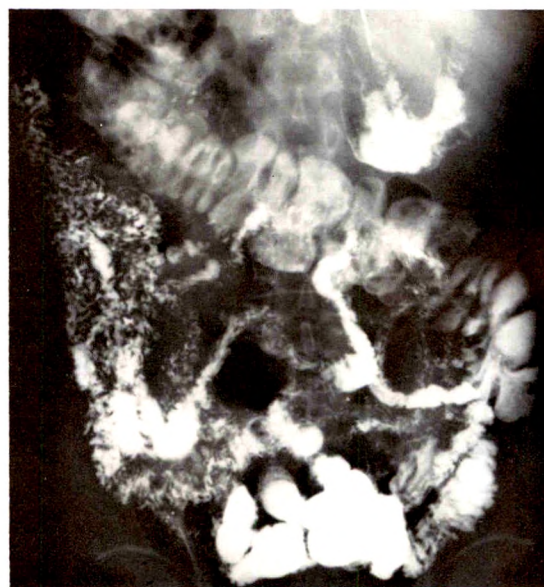


FIG. 3. Ninety minute roentgenogram of small intestine follow-through showing cecum in left lower quadrant and splenic flexure in right upper quadrant.

appears to be the second reported case in the literature and is a mirror image of previously reported cases of inversion of the stomach only. No other congenital anomalies were found in our patient.

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ROENTGEN DIAGNOSIS OF DUODENAL INJURIES

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INJURIES to the duodenum are of two main groups: penetrating injuries and blunt trauma. The former, resulting from stabbing or missiles, are usually explored surgically and are not often seen by the radiologist. When a penetrating injury involves the duodenum, anterior and posterior walls are usually perforated, causing both intraperitoneal and retroperitoneal extravasation. This type of injury will not be discussed further.

The second type of injury, that of blunt trauma to the abdomen, may pose a greater diagnostic problem. In some cases the clinical findings may warrant immediate exploratory laparotomy. In others, it may be difficult to decide whether symptoms and signs are caused by bruising of the anterior abdominal wall or by more serious injury. In such situations, the radiologist may be of considerable help.

Duodenal injury from blunt abdominal trauma is usually manifested by intramural hematoma or retroperitoneal perforation, or by a combination of the two. An intraperitoneal rupture, although common in missile injuries, rarely occurs in blunt trauma. An unexpected blow to the abdomen when the abdominal muscles are relaxed is especially likely to produce injury.

The cases reported here illustrate the roentgen signs that are evident in duodenal injury resulting from blunt trauma.

ANATOMIC FACTORS

Figure 1 illustrates the reason for duodenal vulnerability to blunt upper abdominal trauma. Most of the duodenum lies retroperitoneally, is fixed in position, and unable to avoid an injuring force. The pylorus closes the upper end of the duodenum and the third portion can be com-

pressed over the spine or under the superior mesenteric vessels. Pressure may cause acute angulation and obstruction of the fourth portion. In any of these situations, the duodenum is converted into a closed loop, which, if filled with fluid, further pressure will rupture. If the rupture involves all layers of the duodenum, the contents are extravasated, but if only the muscle layer is burst, an intramural hematoma results.

REVIEW OF THE LITERATURE

In 1954, Felson and Levin³ described the first preoperative roentgen diagnosis of duodenal hematoma. Before that time the diagnosis was established only by surgical exploration or necropsy. Moore and Erlandson¹¹ in 1963 found reports of only 31 duodenal hematomas and described 2 additional such hematomas. If the 2 cases described by Wiot, Weinstein, and Felson¹⁸ are also included, a total of 35 cases is obtained (Table 1).

REPORT OF CASES

CASE 1. A 9 year old boy was admitted on June 20, 1953 with abdominal pain and vomiting of 2 days' duration. On June 17 the boy had fallen against a scooter and bruised his abdomen and lower ribs. He mentioned the injury to his mother at the time but did not appear to be in distress. Medical advice was sought the next day when the child began to vomit and complained of abdominal pain. Examination at the hospital revealed nothing impressive and he was sent home. Vomiting continued on June 19 and abdominal pain progressed throughout the day. Re-examination at the hospital disclosed an upper abdominal swelling and guarding of the right upper quadrant. He was admitted with a clinical diagnosis of possible hemorrhage of the rectus sheath or possible intra-abdominal hemorrhage. He was observed in the hospital during which time plain roentgenograms of the

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abdomen and a gastrointestinal series were obtained (Fig. 2 and 3, A, B and C). At laparotomy,* 7 days after the initial injury, a large intramural hematoma of the second portion of the duodenum was evacuated by incising the muscle. The retroperitoneum around the duodenum was drained via a Penrose drain. No rupture of the duodenum was reported at surgery, but this was undoubtedly present on the roentgenograms. A cardiac arrest complicated the procedure and emergency thoracotomy was required. The patient made a good recovery. A gastrointestinal series 15 days after the operation showed marked improvement with only a small intramural mass remaining.

CASE II. A 28 year old man was involved in an automobile accident in which the upper abdomen was struck by the steering column. Almost immediately severe, generalized abdominal pain was experienced. On examination 1 hour after the accident, diffuse abdominal tenderness was present. No masses were palpable and peristalsis was absent. The abdomen became board-like within 2 hours. Roentgenograms of the abdomen obtained in the interval gave evidence of retroperitoneal gas (Fig. 4). On laparotomy after these roentgen examina-

*The surgical aspects of this case have been reported previously,⁹ but the roentgen findings were not illustrated.

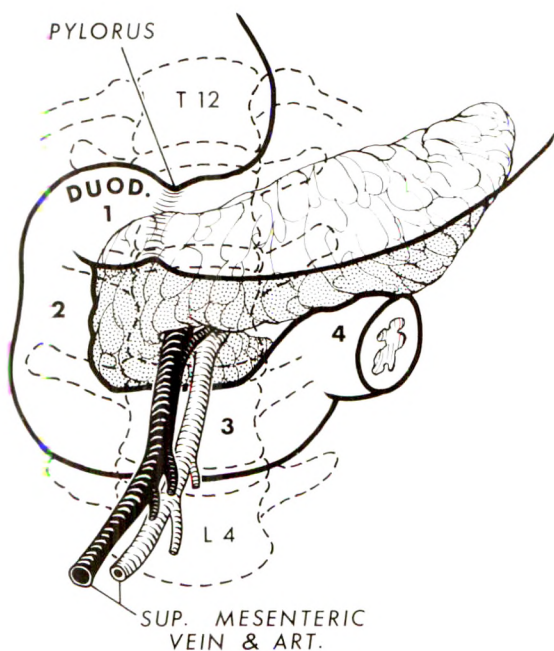


FIG. 1. Diagram of a normal duodenum.

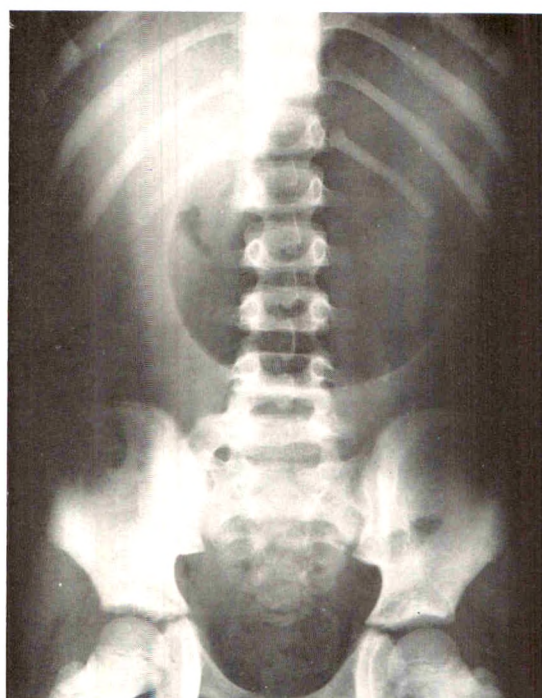


FIG. 2. Case I. Obliteration of the right psoas shadow, distention of the gas-filled stomach, and distortion of the gas-filled duodenum with absence of gas below the duodenum.

tions, a 2.5 cm. retroperitoneal perforation was found in the third portion of the duodenum. The perforation was repaired and a drain left in place. Recovery was satisfactory and the patient was discharged on the thirteenth post-operative day.

CASE III. A 4 year old girl was admitted to the hospital with vomiting of 4 days' duration. The parents evaded further details of the history. Several physicians independently gained the impression that the child had been ill-treated. Both elbows were swollen and roentgenograms revealed healing fractures of both lower humeri. A routine chest roentgenogram

TABLE I

DUODENAL HEMATOMAS REPORTED IN THE LITERATURE

Traumatic	Other	Cause Uncertain	Total
22*	7†	6	35

* Nineteen of these 22 were males; 15 of the 22 were less than 18 years of age.

† Six of these 7 were due to either pancreatic disease or blood abnormality.

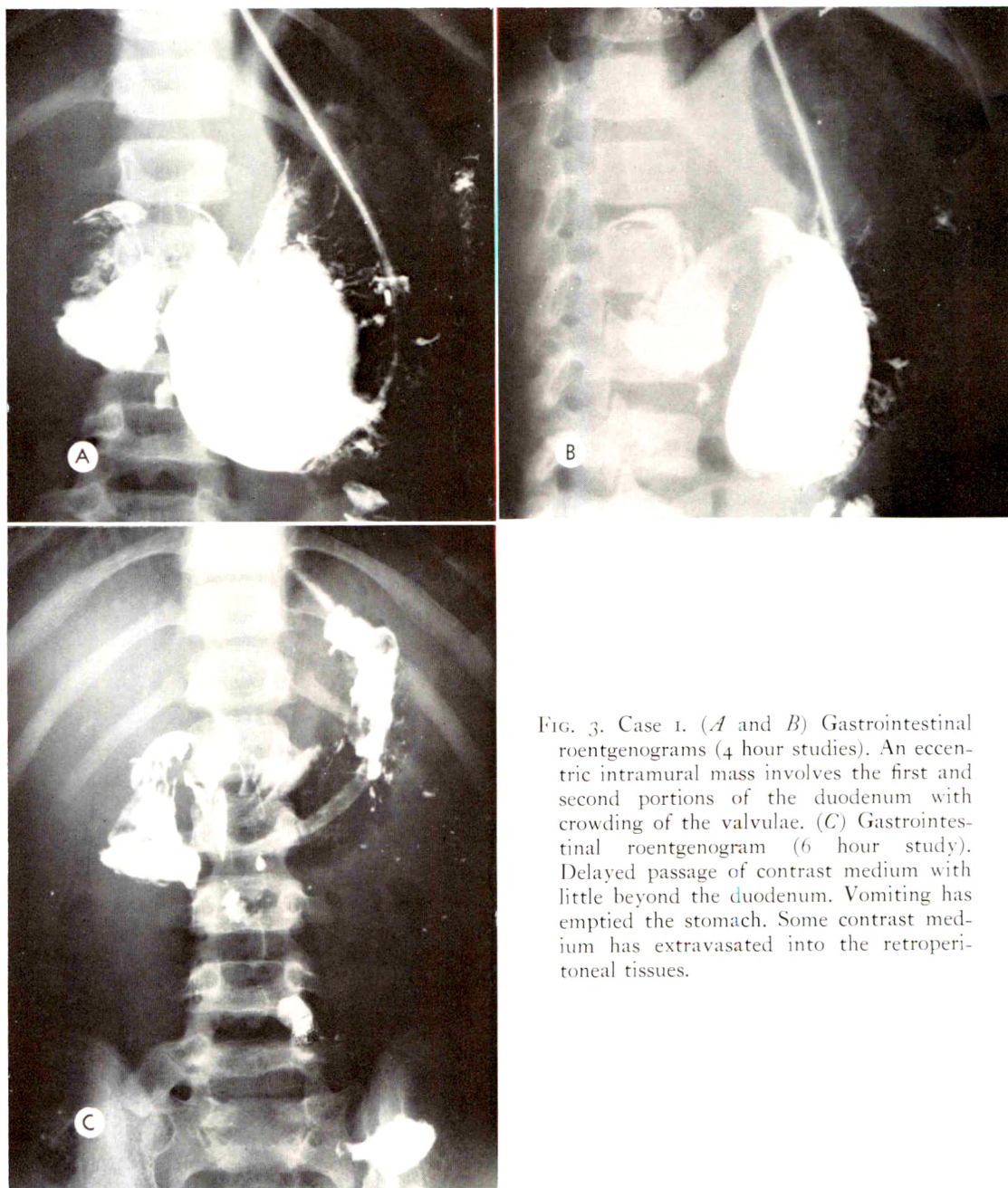


FIG. 3. Case 1. (*A* and *B*) Gastrointestinal roentgenograms (4 hour studies). An eccentric intramural mass involves the first and second portions of the duodenum with crowding of the valvulae. (*C*) Gastrointestinal roentgenogram (6 hour study). Delayed passage of contrast medium with little beyond the duodenum. Vomiting has emptied the stomach. Some contrast medium has extravasated into the retroperitoneal tissues.

showed a healing fracture of the left twelfth rib. The initial examination of the abdomen disclosed a "doughy feeling" but no palpable mass. Peristalsis was diminished but otherwise nothing remarkable was noted. On the fourth hospital day, a 4 cm. by 2 cm. midline upper abdominal mass was felt. A gastrointestinal series disclosed an intramural mass in the second

and third portions of the duodenum (Fig. 5, *A* and *B*).

All abdominal symptoms and signs eventually subsided on conservative therapy. The child was removed from the parents' custody by court order. The diagnosis of duodenal hematoma in this case was based on (a) unequivocal evidence of extensive trauma including ab-

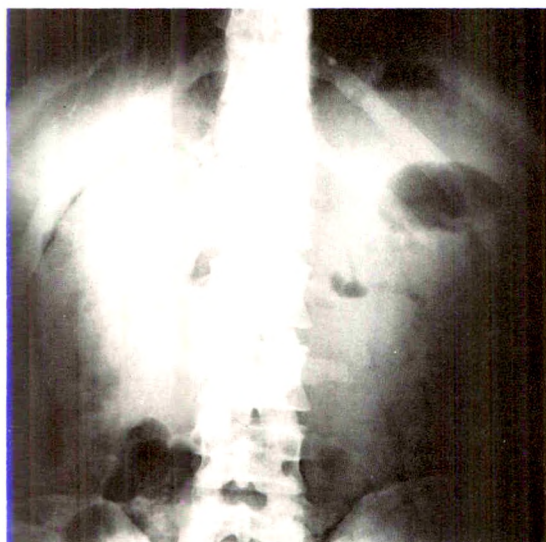


FIG. 4. Case II. Upright abdominal roentgenogram. Retroperitoneal gas outlines the right kidney. No evidence is seen of intraperitoneal gas under the diaphragm. Distended small bowel loops are present in the upper abdomen. The right psoas shadow is obliterated.

dominal trauma (left twelfth rib fracture), (b) a palpable mass in the region of the duodenum, (c) the roentgen findings in the duodenum, and (d) resolution of symptoms and signs on conservative therapy.

DISCUSSION

When blunt trauma to the abdomen results in clinical findings difficult to interpret, plain roentgenograms and studies with contrast medium are important aids to diagnosis. The above described cases and those in the literature¹⁻¹⁷ indicate that, in determining whether blunt trauma has injured the duodenum, the following points should be emphasized.

History. The interval between the time of trauma and the onset of symptoms in duodenal hematoma is frequently from 1½ to 6 days. In duodenal rupture, the onset of symptoms is more rapid and, especially when a large tear is in the duodenum, immediate symptoms can be expected.

Symptoms. Upper abdominal pain and vomiting are the most common.

Clinical Signs. Upper abdominal tenderness, guarding, and rigidity are frequently present. Evidence of external bruising may be present. In duodenal hematoma, a mass may be palpable. The hematocrit value may have dropped.

Roentgen Signs of Duodenal Injury. In duodenal hematoma, the stomach is often dilated by gas. On plain roentgenograms

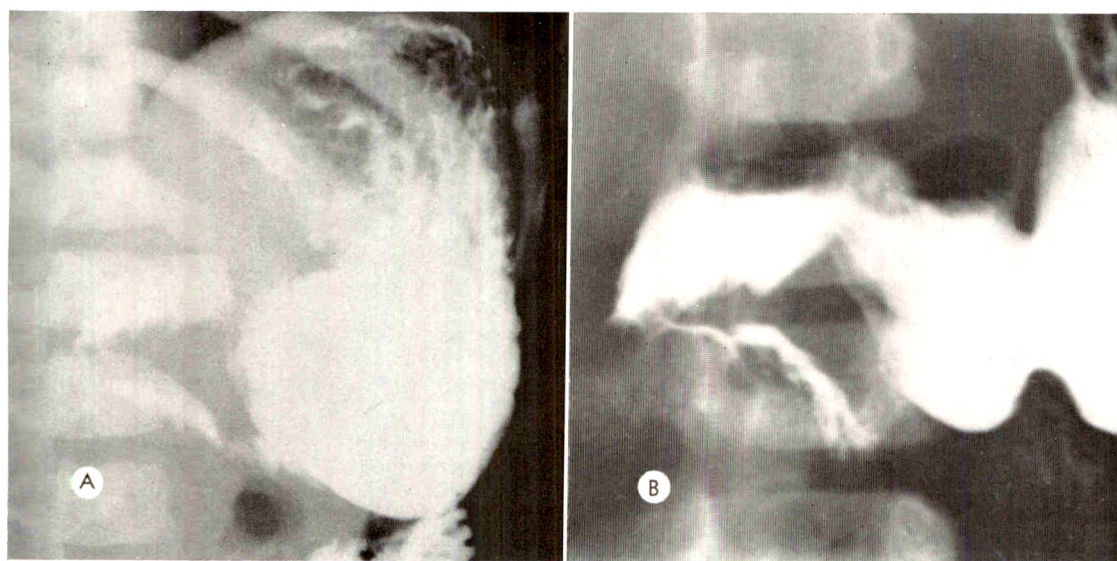


FIG. 5. Case III. (A and B) Gastrointestinal roentgenograms (patient prone and patient upright). An eccentric intramural mass involves the second and third portions of the duodenum. Delayed passage of contrast medium, which, at 6 hours, had all passed.

the hematoma may appear as a mass, impinging on the gastric air-shadow or distorting the gas-filled duodenum. One or both psoas shadows may be obliterated by retroperitoneal hemorrhage. In some cases, dilated small bowel loops may overlie the duodenum, a manifestation of localized ileus. In addition, in duodenal rupture from blunt trauma, retroperitoneal (rarely intraperitoneal) gas may be seen.

Contrast medium by mouth or by a nasogastric tube will show an intramural mass in the duodenum when a hematoma is present. This mass may or may not obstruct the lumen. The hematoma frequently involves a long segment of bowel, a distinguishing feature from other intramural tumors. Some cases of intramural hematoma may show a coiled-spring appearance from crowding of the valvulae conniventes. Felson and Levin⁸ expressed the belief that this coiled-spring appearance was characteristic of a hematoma but the exact cause of this appearance is not known. Presumably, it is related to clot formation in the hematoma as it has not been associated with overdosage of anticoagulants. At first sight, the coiled-spring appearance somewhat resembles that of an intussusception, which, however, in a retroperitoneal portion of the gastrointestinal tract would be unlikely. The frequently eccentric position of the hematoma and the lack of shortening are also distinguishing features.

In duodenal rupture, extravasation of contrast medium (preferably water-soluble) will establish the diagnosis and localize the site.

SUMMARY

The clinical and roentgenologic features of duodenal hematoma and rupture resulting from blunt trauma are described. The mechanisms of injury, clinical history, signs and symptoms, and differential diagnosis are discussed. In the absence of complications or obstruction, some patients with duodenal hematoma, diagnosed roentgenologically, may respond well to conservative

therapy. Follow-up gastrointestinal series in these cases will show that the hematoma has resolved and these patients are spared an unnecessary laparotomy. In others, accurate preoperative diagnosis will shorten the time of anesthesia and surgical procedure.

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The author wishes to thank Dr. William G. Obata, radiologist at Franklin Hospital, San Francisco, for permission to use Case III.

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VILLOUS ADENOMA OF THE DUODENUM

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MANY reports of villous adenoma of the colon and rectum have appeared in recent years. The occurrence of this tumor in the remainder of the gastrointestinal tract, however, has been but rarely reported. In 1962 Moersch *et al.*¹⁰ reported a case of villous adenoma of the duodenum and presented gross anatomic and histologic photographs of that lesion showing the typical findings seen in villous adenoma of the colon or rectum. A recent review of the literature^{1,3,5,9,11} reveals a few cases with lesions having certain resemblances to the lesions reported by Moersch and his co-workers. In the reports, however, microscopic documentation of the nature of these lesions is lacking.

We have studied a patient with histologically proved villous adenoma of the duodenum which was manifest during pregnancy by anemia secondary to persistent gastrointestinal bleeding. In view of the rarity of this tumor in the duodenum, this case is reported.

REPORT OF A CASE

A 25 year old white female was referred to Michael Reese Hospital and Medical Center in the fifth month of her second pregnancy because of profound anemia which could not be accounted for on the basis of expected physiologic changes related to pregnancy. A hemoglobin determination during early pregnancy had been reported as 11 gm. per 100 cc. A hemoglobin determination at the time of admission to the hospital was 5.1 gm. per 100 cc. Oral iron therapy had been given to the patient during the pregnancy.

The patient admitted having vague retrosternal burning after meals and complained of tiredness and weakness. No history of nausea, vomiting or abnormal bowel habits was obtained.

Laboratory investigation, which included

bone marrow study, bleeding, clotting and prothrombin times, platelet count, and a Coombs' test, failed to indicate any primary hematologic disorder. The stools were never free of occult blood (3 to 4 plus benzidine tests). An upper gastrointestinal roentgenologic study was finally performed, using special care to minimize radiation to the fetus. This study revealed a polypoid filling defect in the third portion of the duodenum with trapping of barium within a network of crevices on the surface of the lesion (Fig. 1, A and B).

The patient was given blood transfusions and discharged. She subsequently delivered a viable 4½ pound premature infant. Three weeks later she was re-admitted for surgery. Barium enema examination during this admission showed no abnormality.

At surgery a 4×3.5×1.5 cm. sessile polyp was found in the third portion of the duodenum. Red-pink, papilliferous projections covered the surface of the lesion. There was no evidence of ulceration. The surrounding duodenal mucosa appeared normal. Frozen section and subsequent permanent sections proved the tumor to be a villous adenoma (Fig. 2 and 3) with a morphology identical to large bowel villous adenomas except for the presence of Paneth cells which are normally present in the small intestinal wall. There was no evidence of malignancy. A simple excision of the lesion was performed.

Since the operation the patient has felt well. Hemoglobin studies on different occasions have been between 14.5 and 15 gm. per cent. She became pregnant the following year and had an uneventful gestation with delivery of a normal child.

DISCUSSION

Experience with villous adenomas of the small bowel is very limited; however, much has been written about villous adenomas involving the large bowel. A short, comparative examination of the behavior of

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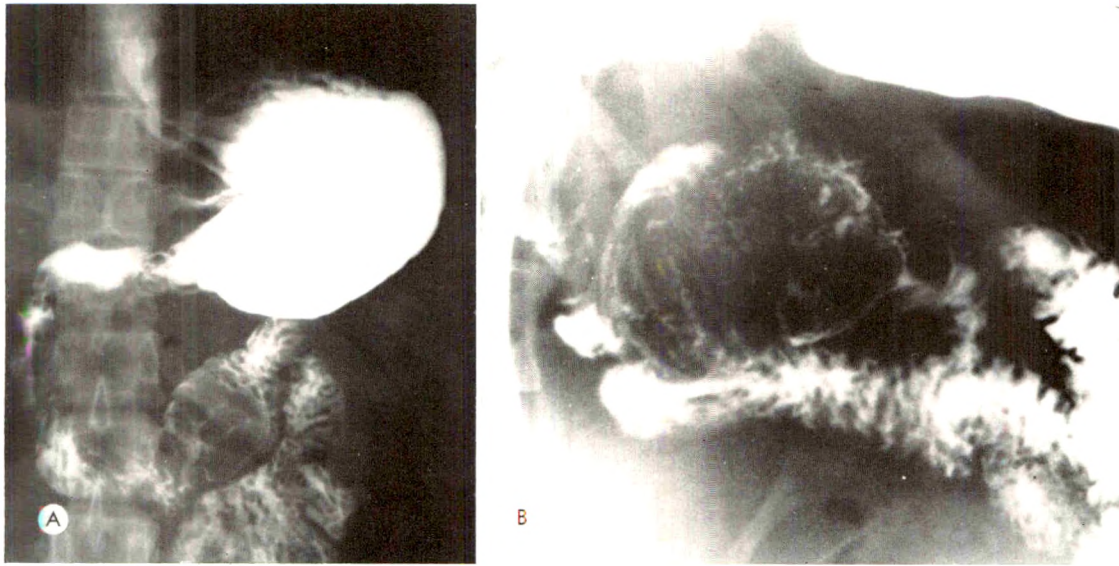


FIG. 1. (A) Prone posteroanterior roentgenogram showing a polypoid lesion expanding the lumen of the third portion of the duodenum. Barium, trapped on the surface of the lesion, forms linear strands in various directions. (B) Compression spot roentgenogram of the lesion, left posterior oblique projection.

these lesions in these two separate locations is of interest.

The incidence of malignancy in large

bowel villous adenomas is high, being most commonly reported as ranging from 31.4 per cent to 74 per cent.^{2,6,13,14,15} The only

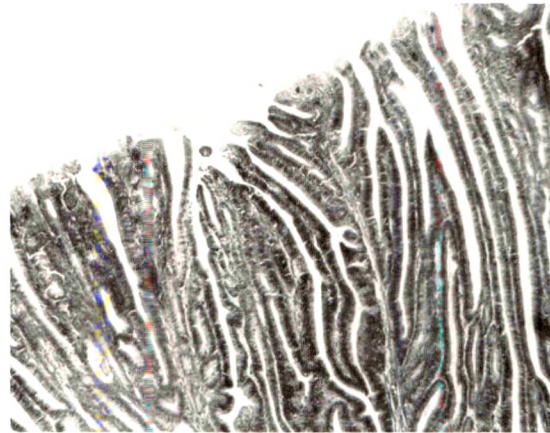
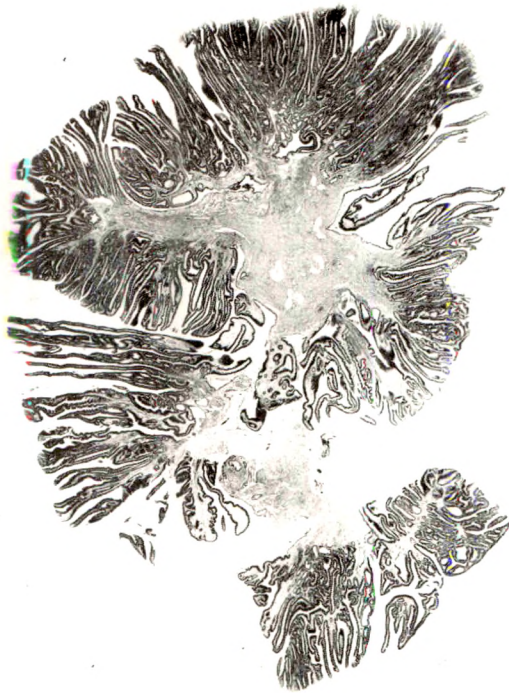


FIG. 3. At higher magnification ($\times 60$) the luminal portion of the papilliferous projections is seen in better detail.



FIG. 2. Low power photomicrograph ($\times 15$) shows long, thin, villous projections arising from a sessile stalk of fibromuscular tissue.

previously well documented case of duodenal villous adenoma proved to be malignant. In our case the tumor was benign.

Bleeding is the most common finding with villous adenomas of the large bowel according to various authors^{8,13,15} with a reported incidence as high as 78.5 per cent.² This appears related to the soft consistency of these tumors and their tendency to fragment and undergo ulceration. Bleeding was not reported as being present in the case of Moersch *et al.* but was the major problem in the case herein reported.

Mucorrhea with large loss of electrolytes has been reported with large bowel villous adenomas,^{4,7,12} a feature responsible for some added interest in this pathologic entity. This finding was not evident in our case nor in that of Moersch *et al.* One may postulate that either these tumors fail to secrete electrolytes or that electrolytes secreted into the proximal bowel become reabsorbed before being lost to the body.

Our case of villous adenoma was first observed in a woman in her fifth month of pregnancy. No association of villous adenomas and pregnancy has been reported. No causal relationship is suggested.

In view of the possibility of malignancy, a preoperative diagnosis of this lesion would be useful. Fortunately, the anatomic configuration of the lesion allows trapping of barium in the crevices around the papilliferous projections on the surface of the lesion. This cauliflower-like appearance encountered in a tumor of the intestinal tract should suggest the possibility of villous adenoma even if the lesion is located in an area other than the large bowel.

SUMMARY

A case of villous adenoma of the duodenum is reported. This is the second fully documented case of this entity. Of interest is its occurrence in a pregnant female. Morphologically, this tumor is almost identical to villous adenoma of the large bowel. Experience thus far suggests that its be-

havior may be similar to the latter's in its propensity to bleed and incidence of malignancy. A preoperative diagnosis should be possible by its characteristic appearance on upper gastrointestinal roentgenologic examination.

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We express our appreciation to Dr. William Thomas, Jr. of the Michael Reese Hospital, Department of Pathology, for his most valuable assistance in the preparation of this report.

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SYNDROME OF JEJUNAL DIVERTICULOSIS AND MEGALOBLASTIC ANEMIA*

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ASSOCIATION of jejunal diverticulosis and megaloblastic anemia is a rare occurrence. The incidence of diverticula of the jejunum has been given as .006 per cent to 1.4 per cent in various autopsy and survey series; it is considerably lower in megaloblastic anemia. Until 1963, only 34 documented cases had been reported in the American and British literature.² Attention has not been directed to this syndrome in the radiologic literature.

This report is prompted by a case of megaloblastic anemia associated with multiple jejunal diverticula which was observed in our hospital recently and which will be described in detail in another publication. We hope to stimulate interest in this bizarre hematologic and gastrointestinal syndrome, for it is the fluoroscopist who, by detecting large jejunal diverticula in a patient known to have anemia, may be able to suggest the diagnosis.

ROENTGEN FINDINGS

The roentgen findings in most of the published cases, including our own, are remarkably similar. The abdominal scout roentgenogram shows scattered pockets of air (Fig. 1). Air fluid levels seen on the upright roentgenogram represent pockets of air entrapped in the large diverticula. This is due to the mouth of the the diverticula being small or twisted, locking the gastrointestinal contents and air within the sac. Several instances are recorded where these air fluid levels erroneously suggested mechanical obstruction of the small bowel on the upright roentgenogram. This mechanism must be considered in the differential diagnosis of small bowel obstruction.^{4,7} Gastrointestinal and small bowel roentgen exami-

nation may reveal many large diverticula of the small bowel, often limited to the jejunum. These may range from small to giant pouches up to 6 or 7 cm. in diameter. In our case, a small hiatus hernia and diverticulosis of the sigmoid colon were interesting associated features (Fig. 2). Difficulty in filling the diverticulum may be encountered because of the aforementioned twisting and stomal narrowing. Considerable technical effort and positioning of the patient may be required to effect flow of the barium into the diverticula.

Stagnation of the contents in the diverti-

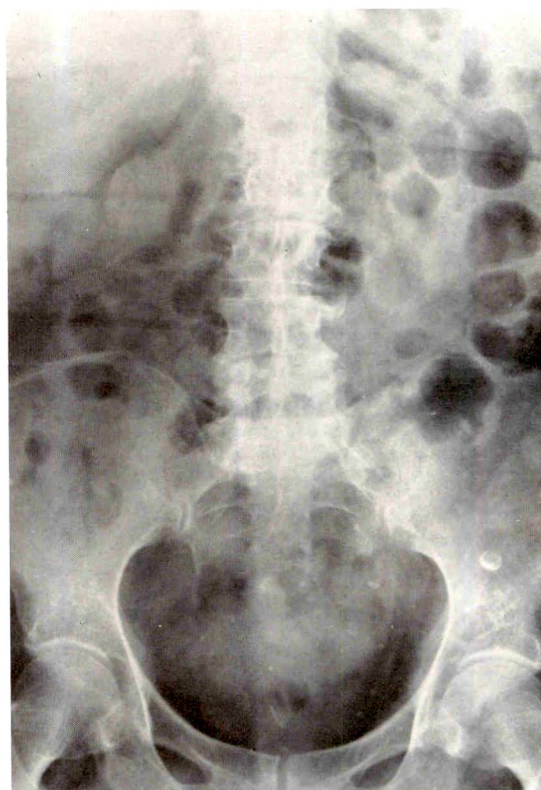


FIG. 1. Abdominal scout roentgenogram. Note multiple air pockets scattered throughout the abdomen representing air in the jejunal diverticula.

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cula has been frequently mentioned as a possible mechanism for the etiology of the anemia. In our case, on roentgenograms taken 4 hours after the administration of barium, the head of the barium column had not reached the colon (Fig. 3). This is considered to represent delayed motor function of the small bowel. Barium may remain in these diverticula and be detected on a 24 hour study.

DISCUSSION

The association of megaloblastic anemias with steatorrhea, diet, sprue, parasitic infestation, blind loops and intestinal anastomoses is well known. Various factors and mechanisms are responsible for the anemia in these conditions, but defective absorption of vitamin B₁₂ is a common finding. In jejunal diverticulosis accompanied by megaloblastic anemia, bacterial overgrowth in the stagnant pouches is be-

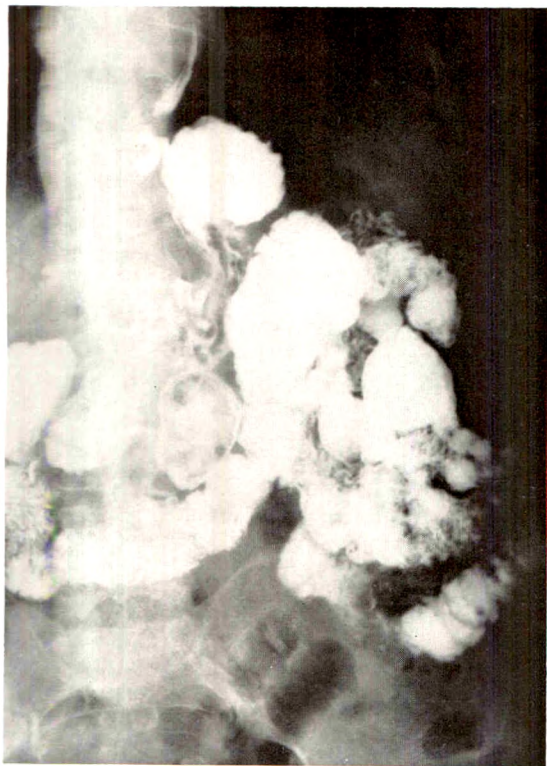


FIG. 2. Barium outlines the stomach and upper small bowel. The large diverticula are easily identified. Also note the small hiatus hernia.

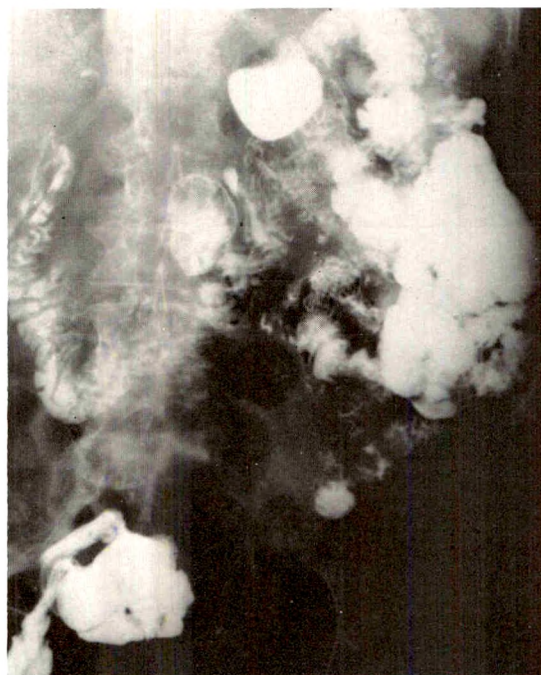


FIG. 3. Roentgenogram made 4 hours after Figure 2. There is slow transit through the small intestine. Several of the diverticula still retain the barium.

lieved responsible for destruction or overutilization of vitamin B₁₂. Proof of this theory is afforded by improvement of the anemia upon administration of antibiotics alone. It is postulated that such therapy will sterilize the infected pouches. Since neomycin may be responsible for, or aggravate, the malabsorption syndrome, its use is contraindicated in these cases.³ Others have been cured by segmental resection of the jejunum containing the diverticula followed by end-to-end anastomosis.^{5,6} A similar situation is known to exist in infestation with the fish tapeworm, *Diphyllobothrium latum*. The latter utilizes the available vitamin B₁₂. Treatment accordingly is directed to the removal of the parasite.

Many of the reported cases have shown free stomach acid, thus ruling out pernicious anemia. In others, however, the presence of achlorhydria has led to a problem in differential diagnosis. The exact relationship of achlorhydria, pernicious anemia and defective vitamin B₁₂ absorption due to

TABLE I
VITAMIN B₁₂ ABSORPTION IN THE DIFFERENTIAL DIAGNOSIS OF MEGALOBlastic ANEMIAS

Test	Pernicious Anemia	Jejunal Diverticulosis	Sprue
Radioactive B ₁₂ alone	Poor	Poor	Poor
Radioactive B ₁₂ plus intrinsic factor	Improved	Poor	Poor
Radioactive B ₁₂ plus antibiotic	Poor	Improved	Poor

the small bowel diverticula is unknown. Differential diagnosis, however, can be made (1) by using the Schilling test with radioactive B₁₂ alone, (2) radioactive B₁₂ plus intrinsic factor, and (3) radioactive B₁₂ plus an antibiotic (Table I). In pernicious anemia there will be an improvement when intrinsic factor is added and little improvement with the other two tests. In the jejunal diverticula or blind loop syndrome, improvement will only be noted when the radioactive vitamin B₁₂ plus the antibiotic test is used. In other cases of steatorrhea and megaloblastic anemia, such as sprue, there is a poor response to all three tests. It should be noted that in all published cases to date a megaloblastic bone marrow has been found.

Malabsorption syndrome, usually mild or moderate, has also been noted in most of the patients with jejunal diverticula. Several explanations for this have been advanced, and of these loss of absorption surface by the thin-walled diverticula, altered motility, and mechanical interference with lymphatic flow seem most plausible. Others have suggested that stasis and bacterial interference with fat absorption may produce a steatorrhea-like picture.^{1,6} The role of folic acid in this entity is of some doubt. The jejunum is reported to be the major site of folic acid absorption, yet in the reported cases only one showed improvement when treated with folic acid alone.⁷ The intricate relationship between vitamin B₁₂ and folic acid metabolism may furnish a clue to the failure of folic acid to produce a

cure in the absence of B₁₂. Such therapy is also ineffective in treating subacute combined degeneration and pernicious anemia.

The rarity of this disease may only reflect a lack of appreciation of the causal relationship between hematologic and roentgenographic findings in this entity. Such knowledge, therefore, concerning jejunal diverticulosis associated with megaloblastic anemia should enable the radiologist to suggest this diagnosis with greater frequency.

SUMMARY

1. A brief review of the hematologic picture and etiology of megaloblastic anemia associated with jejunal diverticulosis is presented.
2. The roentgen findings of this syndrome are described and should alert the radiologist to this rare combination.

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SMALL BOWEL OBSTRUCTION DUE TO AN UNUSUAL VERMIFORM MECKEL'S DIVERTICULUM DEMONSTRATED BY BARIUM ENEMA STUDY*

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A MECKEL's diverticulum was recently demonstrated on a barium enema study in a patient with an acute small bowel obstruction. The unusual appearance of the diverticulum and the absence in the English literature of this finding in an acutely obstructed patient have prompted the description of this case.

REPORT OF A CASE

A 22 year old white male student was admitted to the UCLA Medical Center with a 5 day history of constipation, obstipation, mild epigastric distress, and postprandial emesis. His past history was unremarkable.

The vital signs were within normal limits and he was afebrile. The abdomen was moderately distended, nontender, quiet and without masses or organomegaly. Rectal examination revealed no masses or tenderness and sigmoidoscopy was negative to 10 inches.

The hematocrit was 50 per cent and the white blood cell count 7,500 with a normal differential. Abdominal roentgenograms demonstrated generalized small bowel distention with multiple air-fluid levels in the upright and decubitus positions (Fig. 1). A moderate amount of fecal material was noted in a nondilated cecum and ascending colon. Because of the possibility of a bowel obstruction, a barium enema examination was performed. The colon was normal and the appendix filled out well. During the procedure, barium refluxed into the ileum with filling of a long narrow segment of bowel measuring 0.3 by 6.5 cm. Fluoroscopically, the structure was freely movable and only slightly tender to palpation (Fig. 2, *A* and *B*). It was felt that this represented the site of the obstruction. Although a Meckel's diverticulum was considered, this diagnosis was not definitely made because of its atypical appearance.

Subsequent abdominal exploration 3 hours later revealed a long narrow Meckel's diverticu-

lum arising from the antimesenteric border of the ileum about 50 cm. from the ileocecal valve (Fig. 3). Its tip was adherent to the small bowel mesentery. In rotating more than 180° and attaching to the mesentery, the diverticulum twisted the ileum and caused the obstruction. A diverticulectomy and appendectomy were performed. Postoperatively, the patient did well except for a moderately prolonged paralytic ileus.

DISCUSSION

It is well known that a Meckel's diverticulum is a common anomaly with an incidence range from 1.1 to 4.5 per cent in the various reported series.¹⁷ This structure oc-

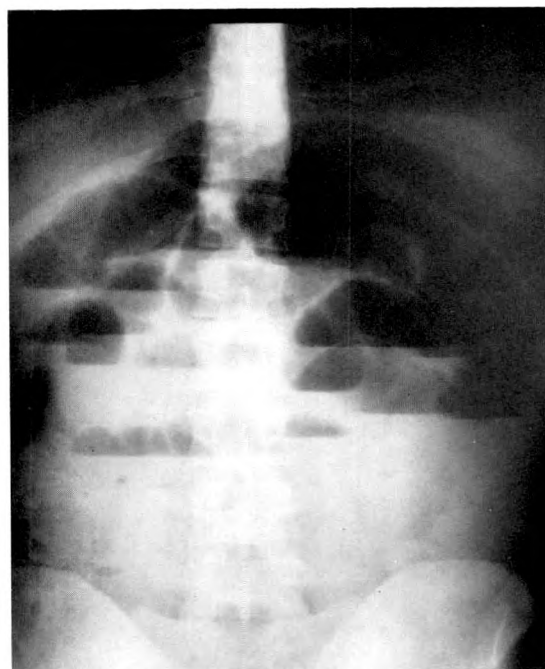


FIG. 1. Upright roentgenogram of the abdomen showing small bowel distention with multiple air-fluid levels.

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curs when the omphalomesenteric duct connecting the yolk sac with the intestinal tract fails to completely obliterate.^{10,21} It originates from the antimesenteric border of the ileum at a variable distance from the ileocecal valve and has an independent blood supply.^{11,17} It contains the normal layers of small intestine, which is suggested fluoroscopically if peristaltic activity is discerned.^{11,12} The appearance and size of a Meckel's diverticulum vary considerably. Usually, they have the configuration of a "finger-like" pouch with a broad base. Occasionally, however, vermiform diverticula have been found during surgery.¹⁷ In a review of the literature, there is no re-

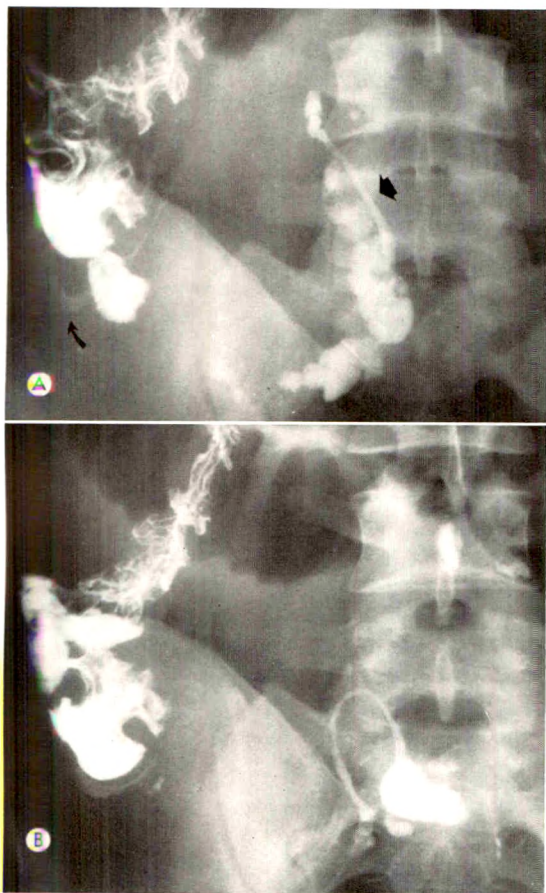


FIG. 2. (A) Postevacuation roentgenogram during barium enema study showing a long "appendix-like" Meckel's diverticulum (arrow). Note that there is separate filling of the appendix (curved arrow). (B) Subsequent roentgenogram showing motility of the diverticulum.

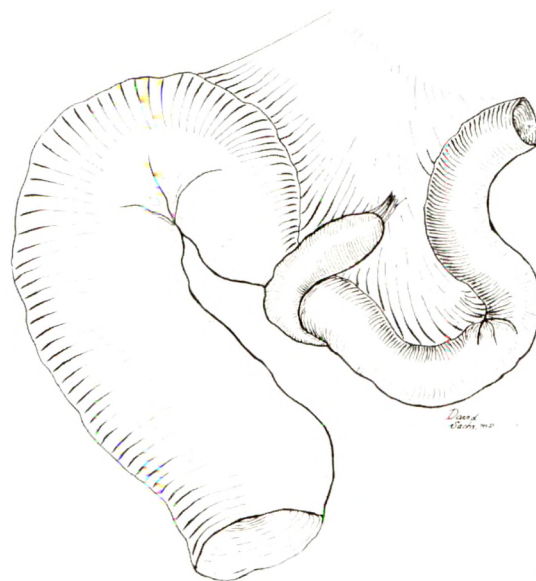


FIG. 3. Diagrammatic representation of operative findings in which a Meckel's diverticulum was causing a small bowel obstruction.

port of an "appendix-like" Meckel's diverticulum demonstrated either by barium meal or barium enema examination.

The roentgenographic demonstration of a Meckel's diverticulum is unusual and is frequently discovered incidentally. It is felt by some that lack of visualization of the diverticulum results from a wide mouth and active persistalsis, making retention of barium difficult.^{3,7} Overlying bowel loops filled with barium add to the difficulties of identification.

Meckel's diverticula have been demonstrated most often during the course of a small bowel study.¹⁷ Certain authors, however, feel that they are easier to demonstrate by reflux of barium into the ileum during a barium enema examination.^{1,13}

Bischoff and Stampfli⁴ reviewed the literature in 1955 and noted 8 cases in which the diverticulum was demonstrated by a barium enema study; the first was reported by Prévôt¹⁵ in 1936. Fifteen additional cases were uncovered in the English literature.^{1-3,5,6,8,9,11,13,14,16,18,20} In none of these 23 cases was the examination per-

formed in the face of an acute obstruction. These studies were most often done on patients with chronic intestinal bleeding or abdominal pain.

Intestinal obstruction caused by a Meckel's diverticulum is not uncommon. Veith and Botsford¹⁹ found that in a series of 1,810 cases with complications, 23 per cent were due to intestinal obstruction, including intussusception.

Whereas hemorrhage from peptic ulceration in a Meckel's diverticulum is seen more often in children; obstruction excluding that due to intussusception in children, is seen in patients of all ages.^{17,19,21} Bowel obstruction due to a Meckel's diverticulum can be caused by inflammation, intussusception, band formation from diverticulum to small bowel mesentery or intestinal wall or twisting of the bowel around the diverticulum.

A barium enema examination in an obstructed patient is considered unnecessary by some physicians when there is an established indication for abdominal exploration. Occasionally, however, the site of obstruction can be established preoperatively and the organic pathology clarified by use of the barium enema study. Moreover, as in the case presented, the barium enema examination was a significant aid in confirming the diagnosis of mechanical small bowel obstruction.

SUMMARY

A case is presented in which an unusual vermiform Meckel's diverticulum was demonstrated on a barium enema study in an acutely obstructed patient. A similar report could not be found in the English literature, nor has a Meckel's diverticulum with a configuration similar to the appendix demonstrated by barium examination been reported. It is well recognized that its roentgenographic demonstration is difficult and uncommon.

Although Meckel's diverticula have been diagnosed more frequently during a small bowel study, it is suggested that their

visualization is more easily accomplished if barium can be made to reflux into the ileum during an enema examination.

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THE MASS SIGN IN PRIMARY VOLVULUS OF THE SMALL INTESTINE IN ADULTS

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IT IS generally accepted that the pre-operative diagnosis of volvulus of the small bowel is extremely difficult, particularly from the roentgenologic standpoint. In view of the necessity of prompt surgical intervention, we feel that the roentgenographic criteria we have encountered in 3 cases of primary volvulus of the small bowel are helpful in establishing an early diagnosis. Although the pseudotumor sign of Frimann-Dahl⁴ has been described in closed loop obstruction, simple obstructions, and volvulus, the presence of a definite mass with a sharp, smooth border in primary volvulus has not been stressed. Williams,¹⁵ and Frimann-Dahl⁴ each show an example of a volvulus with a mass noted on the roentgenogram. It is the purpose of the authors to call attention to the mass lesion in primary volvulus of the small intestine and to discuss briefly the differential diagnosis. With the clinical history in focus, the radiologist should, we feel, be able to suggest the correct diagnosis.

Volvulus of the small bowel refers to torsion or rotation of a segment of small intestine resulting in obstruction. The occlusion of the lumen occurs at both the proximal and distal ends of the involved loop at the fixed point of rotation. Thus, there is obstruction proximal to the involved segment, which itself is transformed into a closed loop. The obstruction of the bowel due to volvulus may be complete or incomplete, although usually the former obtains.⁶

Lulenski⁷ differentiates between primary and secondary volvulus of the small bowel. In the primary volvulus simultaneous torsion, closed-loop formation, and simple obstruction proximally are present. Adhesions are the most common etiologic factor. A

long mesentery^{2,8} or a short mesenteric root⁶ predisposes to primary small bowel volvulus.

Alternatively, the volvulus may be secondary to pre-existing intestinal obstruction of 24 hours', or more, duration. This is caused by violent peristalsis of the movable bowel with secondary volvulation.^{5,12}

Other causes of primary or secondary volvulus are Meckel's diverticulum, internal hernia, tumors, foreign body impaction, vascular occlusion, and developmental anomalies of rotation.^{2,3,10}

CLINICAL PATHOLOGY

Severe, crampy abdominal pain and vomiting are the usual presenting symptoms.⁶ Shock may occur secondary to venous engorgement and venous pooling or arterial obstruction in the splanchnic bed.⁸ Perforation may occur with resulting peritonitis. A mass may be palpable either through the anterior abdominal wall or on vaginal examination.¹⁴

It should be stressed that the more severe the twist, the greater the incidence of interference with the blood supply and the possibility of ensuing gangrene. Venous occlusion followed by arterial obstruction is the usual sequence. If operative intervention is carried out early enough, the pathologic process may be reversed by simple untwisting of the volvulus without the necessity of resection.^{1,2}

ROENTGEN FINDINGS

In small bowel volvulus secondary to previously existing obstruction, the roentgen findings may be entirely nonspecific because the volvulated loops had previ-

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ously been distended with air. In primary volvulus, on the other hand, an early sign may be a density in the abdomen which has the appearance of a soft tissue mass. This would fit under one cause of "pseudotumor" described by Frimann-Dahl, and is considered pathognomonic of strangulation.⁴ The size of this density varies with the amount of bowel involved in the torsion.

Some roentgenographic features considered characteristic of small bowel volvulus (primary and/or secondary) are a whorled, polycyclic appearance of the gas filled small bowel loops; widely scattered air-fluid levels in the upright or decubitus position (the fluid levels are of larger size than simple obstruction—the large bowel is not distended); the separation of the bowel loops by free intraperitoneal fluid which produces pelvic opacity in the upright position; and bands of density outlined by radiolucencies due to air between thickened folds of fluid filled small bowel.^{4,11}

An isolated, gas distended small bowel loop in the abdomen should also bring volvulus to mind. Such a loop with its segments separated by thickened intestinal walls produces the "coffee bean" appearance of Mellins and Rigler.⁸ In the presence of free intraperitoneal air, such a dilated small bowel loop suggests the presence of a gangrenous small bowel loop which has perforated, the gangrene having been produced by the volvulus.

Pneumoperitoneography was suggested as a special procedure which would help in making a more specific diagnosis. In 1956, Perry, Von Drashek, and Wangenstein⁹ injected 500–1,000 cc. of air into the peritoneal cavity when strangulation of a loop of a small bowel was suspected. They noted that the increased amount of blood in the affected small bowel lumen and its wall caused it to be increased in density. As a result, the small bowel loop stood out clearly against the background of air in the remainder of the abdominal cavity.

The use of an opaque medium has been advocated in cases where the roentgen find-

ings are few, particularly when strangulation has occurred. Frimann-Dahl⁴ and Williams¹⁵ have recommended the use of barium sulfate. Vest¹³ has used a soluble opaque medium to differentiate between simple obstruction and strangulation. He noticed that the opaque medium in a simple obstruction arrived at the obstructed point in approximately 1 hour. In the presence of strangulation, however, the stomach emptied poorly, thus suggesting strangulated small bowel.

Barium enema studies in cases of doubt serve to rule out colonic obstruction and dilatation. This is especially helpful when the small bowel becomes markedly distended with absence of folds due to gangrene so that it simulates the colon.⁴

The above criteria, while suggestive of volvulus, need not be pathognomonic and may be encountered in other types of small bowel obstruction.

REPORT OF CASES

CASE I. J.D., Boston City Hospital #2015101. This 22 year old white male entered the hospital on July 7, 1964, with a 2 day history of abdominal and back pain. There was associated vomiting, 20 to 30 times per day.

Past history revealed chest trauma on the left side, with multiple healed rib fractures.

Physical Examination. The abdomen was tense, but no specific mass could be felt. Bowel sounds were hyperactive initially.

Laboratory Data. The white blood cell count was only slightly elevated (12,000).

Roentgen Findings. A plain roentgenogram of the abdomen showed a soft tissue mass in the upper mid abdomen (Fig. 1). A barium study showed acute obstruction with torsion of the duodenal folds proximal to the ligament of Treitz (Fig. 2). A loop of bowel having the appearance of colon was noted above the left hemidiaphragm.

Surgery. At surgery, 64 cm. of small bowel, extending from the ligament of Treitz distally, was gangrenous. This loop of bowel had undergone volvulus, which was complete. It was also noted that the left colon had herniated through a defect in the left hemidiaphragm (presumably from old trauma), and a left hemicolectomy was done because the herniation

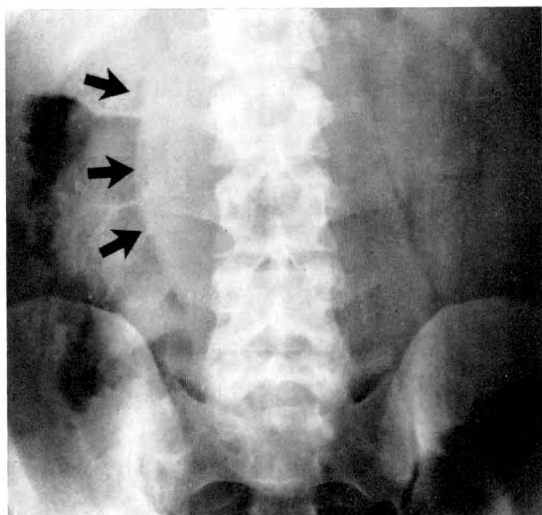


FIG. 1. Case I. Plain roentgenogram of the abdomen showing a large mass with a sharp smooth right border (arrows).

had irreversibly compromised the blood supply to the left colon. The patient did well and was discharged in good condition.

CASE II. F.W., Boston City Hospital #1815190. This 16 year old white male entered the hospital on May 11, 1964, with abdominal pain of 3 days' duration. No back pain or significant emesis was noted.

Physical Examination. Minimal tenderness was present, but bowel sounds were normal. No mass was felt.

Laboratory Data. The white blood cell count was 12,000.

Family History. One sibling had cystic fibrosis.

Hospital Course. In the evening of the same day, the patient went into shock, and the white blood cell count rose to 20,000.

Roentgen Findings. Plain roentgenogram of the abdomen showed a soft tissue mass in the right lower quadrant (Fig. 3), with small amounts of air in the small and large intestine. Upright study showed minimal large and small air fluid levels with no free air.

Surgery. The entire small bowel from the ligament of Treitz down to the ileocecal valve had undergone acute volvulus with resultant gangrenous bowel. The duodenum, a small segment of jejunum, and a few centimeters of terminal ileum were left in place, with appropriate anastomosis. It was noted that the

mesentery was extremely long and was probably the etiologic factor. The patient did well.

CASE III. J.I., an 18 year old white male, was admitted complaining of intermittent abdominal and back pain of 12 hours' duration. There was associated vomiting. These symptoms began while the patient was participating in a football game.

Physical Examination. There was marked tenderness over the entire abdomen, especially in both upper quadrants. No mass was felt.

Laboratory Data. The white blood cell count was 41,200.

Roentgen Findings. A plain roentgenogram of the abdomen showed a large soft tissue mass in the mid abdomen extending up to the level of the epigastrium. There was some displacement upwards of the transverse colon (Fig. 4).

Surgery. A 180 degree volvulus of the small bowel extending from a point 6 inches distal to the ligament of Treitz to a point 24 inches proximal to the ileocecal valve was found. Arterial pulsations were noted in the mesenteric vessels. Serosanguineous fluid was aspirated from the peritoneal cavity. The volvulus was untwisted, and the previous purplish color regressed. No resection was performed. The patient subsequently went into shock and expired a few hours after the operation.

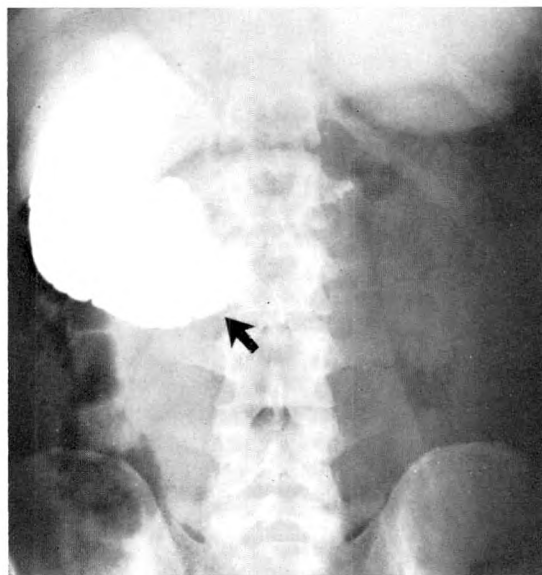


FIG. 2. Case I. Barium study shows torsion of the folds with obstruction proximal to the ligament of Treitz (arrow).

DISCUSSION AND DIFFERENTIAL DIAGNOSIS

In 3 cases of primary volvulus of the small bowel, the mass of water density was particularly well shown. With the clinical history of sudden abdominal pain, emesis, and possibly shock, correlative diagnosis can be made. In one case, the use of an opaque medium served to outline the point of obstruction in the distal duodenum with the characteristic tapering indicative of torsion.

In differential diagnosis, one must consider simple small bowel obstruction, internal hernia, vascular occlusion, and the so-called "sentinel-loop" associated with an inflammatory process in an abdominal viscus.

If the loops of small bowel are markedly distended, a simple small bowel obstruction may be simulated. In this instance, the criteria listed under roentgen diagnosis may be helpful in differentiation.

When the mass-like density is demonstrable roentgenographically, the possibility of internal hernia or a vascular oc-

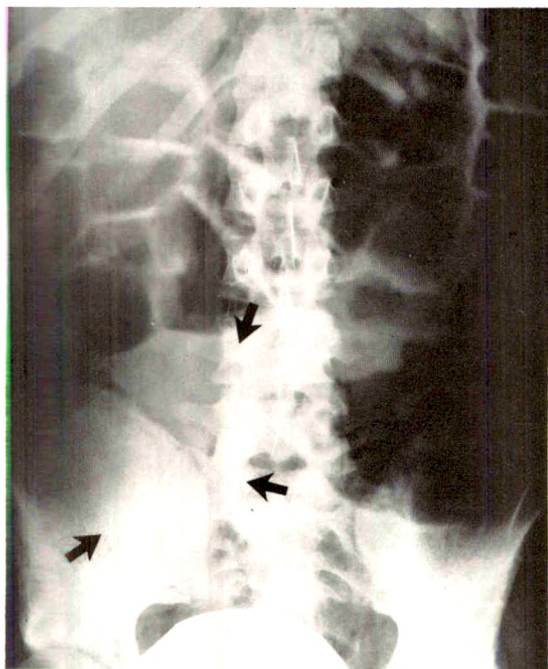


FIG. 3. Case II. Plain roentgenogram of the abdomen shows well defined ovoid mass in the right lower quadrant (arrows).

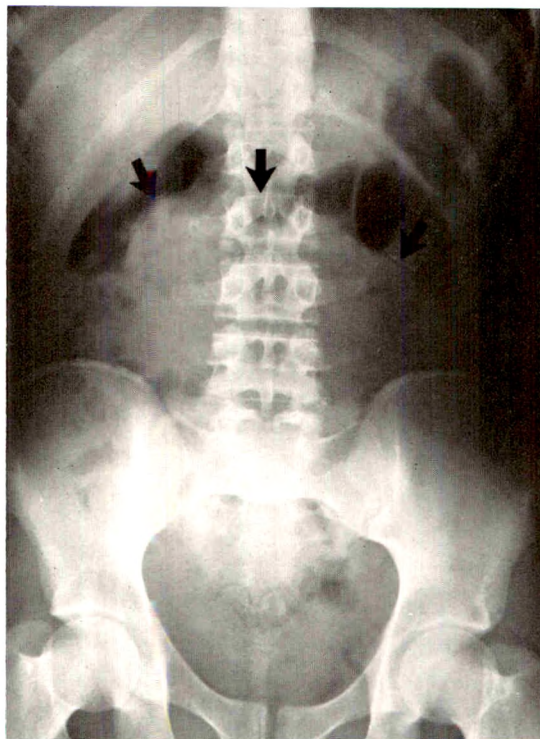


FIG. 4. Case III. Plain roentgenogram of the abdomen shows a huge mass in the mid abdomen extending up to the level of L₁ (arrows). Some superior displacement of the transverse colon is present. The gas distribution is almost exclusively of the large bowel pattern.

clusion must first be ruled out. The latter is especially true in the older age group or in the presence of cardiac valvular disease. An acute exacerbation of chronic pancreatitis with a pseudocyst should also be considered.

In the presence of a single dilated loop of small bowel in an acute abdomen, the possibility that this represents a sentinel loop with an inflammatory process such as pancreatitis, cholecystitis, or appendicitis may be brought to mind. These can usually be ruled out on clinical grounds.

Simple obstruction and acute pancreatitis with pseudocyst can usually be ruled out on the basis of clinical and historical criteria. Vascular occlusive disease is extremely difficult to differentiate and we feel that arteriographic studies, if clinically feasible, are necessary in some cases for a clear diagnosis. Strangulation of an internal

hernia with the formation of a mass could mimic volvulus.

CONCLUSION

In the presence of acute abdominal pain, vomiting, possibly shock, with or without a palpable abdominal mass, the roentgenographic finding of a mass in the abdomen should alert the radiologist to the strong possibility of primary volvulus of the small bowel. Serial radiopaque examination of the small bowel may be helpful when the plain roentgenographic findings are minimal.

SUMMARY

Three cases of primary volvulus of the small bowel are reported. With the clinical history and the demonstration of a mass in the abdomen, the probability of primary volvulus of the small bowel must be considered. Characteristically, the mass is well defined with a sharp smooth border.

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RECOGNITION OF ASCARIASIS BY ROUTINE CHEST OR ABDOMEN ROENTGENOGRAMS*

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ONE of the most common intestinal parasites²⁹ is *Ascaris lumbricoides*. It was first described in Eber's papyrus dated about 1550 B.C.¹¹ and continues to cause symptoms, illness, and even death^{16,26} to this day. It is by far most common in the tropics and subtropics,¹² but has also been found in the cooler climates of the world.^{7,17,22,25} The life cycle of this parasite has been well established,^{6,27} and effective therapy for eradication and prevention is available.

Improved therapeutic agents,⁹ increased awareness of the multitude of symptoms that can occur,^{30,31} and better sanitary measures have noticeably decreased the over-all incidence of ascariasis in this country; however, the warmer portions of the United States remain a stronghold, especially in the lower socio-economic groups.¹²

With the extensive migration of the population to new and different parts of the country, it is expected that more cases will be seen in areas in which it has heretofore been unusual. It is therefore felt that physicians should be made more aware of the findings that are common on plain roentgenograms of the chest and abdomen in this disease entity, and of the signs that will lead to a diagnosis of or suspicion of ascariasis as a cause of symptoms.

HISTORICAL REVIEW

In 1922 Fritz⁸ first described the picture of small bowel Nematodes from barium contrast studies. Since then, many reports have appeared.^{2,21,34} *Ascaris* worms have been demonstrated in all parts of the bowel, stomach, bile ducts, pancreas, pleural cavities, bladder, fallopian tubes, brain, and eye.²⁶

Lenarduzzi,¹⁹ in 1938, was the first to visualize intestinal ascariasis without the use of contrast agents. Other reports^{1,3,4,10} appeared in the European literature during the war years but were slow to reach the rest of the world.

Skapinker,²⁸ in 1949, demonstrated on plain roentgenograms *ascaris* worms outlined by gas in the jejunum in a patient with intestinal obstruction, and was the first to report this in the English literature. Other articles followed,^{15,23} with the papers of Isaacs¹³ and Daurelle⁵ containing the best illustrations.

MATERIAL

All cases coded as ascariasis at Charity Hospital, New Orleans, between July 1, 1961 and May 13, 1964 were reviewed. These included only the patients who were hospitalized, not the many cases seen and treated in the outpatient clinics. Cases in which the diagnosis of ascariasis was primary as well as incidental to other problems were included. The roentgenograms made from the time of diagnosis, whenever available, were then examined. They included studies of the chest only, abdomen only, or both.

RESULTS

Four hundred and fifty seven cases of ascariasis were diagnosed at some time during the patient's hospital stay. In 262, roentgenograms were available for review. These were divided into 2 groups: the normal, inadequate and not helpful in diagnosis, which were excluded, and the abnormal roentgenograms which are the basis of this report. Several cases were excluded from

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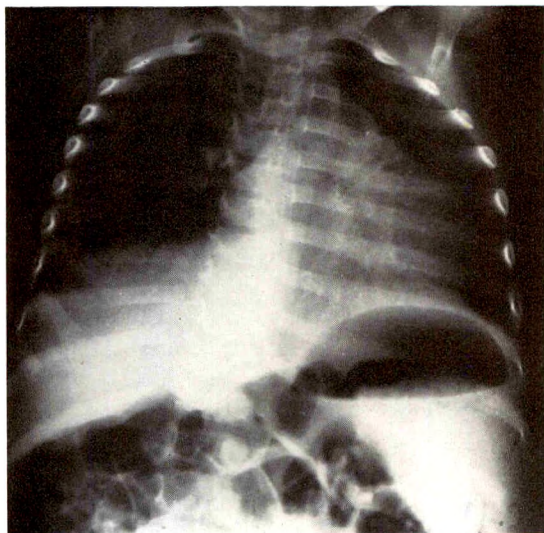


FIG. 1. This erect posteroanterior roentgenogram of the chest included enough of the abdomen to visualize multiple rounded opacities in the small bowel of the left abdomen. An eosinophil count of 26 per cent and a bilateral bronchopneumonia (not well seen here) indicate a reinfection with *Ascaris lumbricoides*. Adult worms are present in the bowel and larvae were aspirated from the stomach.

the study because of inadequate clinical proof even though the diagnosis was obvious from the roentgenograms.

Roentgenographic correlation of ascariasis was possible in 95 proved cases. Seventy-eight of these showed definite evidence of ascaris worms on plain roentgenograms, 6 cases demonstrated the worms on contrast studies and 11 patients showed signs that were not conclusively diagnostic. In 40 of the 78 cases with definite roentgenographic evidence, the ascaris could be identified on either an erect posteroanterior (Fig. 1) or lateral (Fig. 2) study of the chest, or both. In the remaining 38, flat and/or erect roentgenograms of the abdomen were necessary for the diagnosis.

The age range of the patients was from 10 months to 15 years with 84.5 per cent being 6 years of age or less. An 11 year old and the only case older than 12 years of age (15 years) were mentally retarded.

In 81 cases symptoms were adequately recorded for evaluation. Fifty-two patients, or 64 per cent, had symptoms refer-

able to the ascaris infestation in addition to any nonrelated symptoms. Twenty-nine patients, or 36 per cent, were free of symptoms that could be related to the parasite. In this latter group ascariasis was usually an incidental finding.

Differential blood counts were available on 78 patients. Twenty, or 25 per cent, were considered to have no eosinophilia or less than 1 per cent. Forty patients (51 per cent) had eosinophil counts greater than 3 per cent and 20, or 25 per cent, greater than 10 per cent.

Twenty-four patients out of the 95 (25 per cent) demonstrated pneumonia on chest roentgenograms, varying from a typical Loeffler's type^{20,31} (Fig. 3) to a small localized area of infiltration. It was interesting to note that the percentage of patients with pneumonia was the same as that of patients with an eosinophil count greater than 10 per cent. Although there was not complete correlation between these two findings, the occurrence is not due to chance alone. Two patients with an eosino-

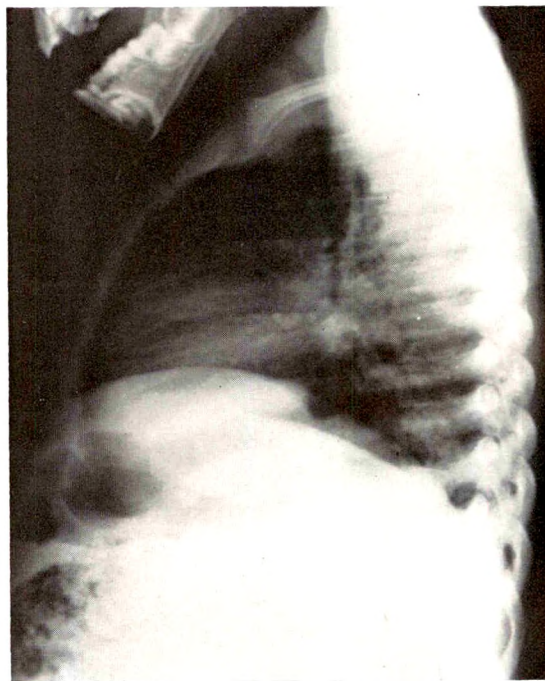


FIG. 2. A lateral roentgenogram of the chest demonstrates rounded opacities in the anterior abdomen, indicating ascaris worms in the axial projection.

phil count of 12 and 21 per cent, respectively, were free of pneumonia, but this can be expected occasionally.¹⁸

The degree of infestation did not always coincide with the symptoms. One 11 year old girl was admitted for evaluation of lymphedema of one foot. Massive ascaris infestation was incidentally noted on the intravenous pyelogram (Fig. 4). Another child, 2 years old, was seen with dark yellow urine, swollen hands and knees, a running nose, rhonchi, and dullness in the lung bases. He was known to be a chronic dirt eater. He was afebrile and had a white blood cell count of 31,400 with 26 per cent eosinophils. Stool examination was negative and a liver biopsy was normal. Sick cell preparations were negative and virus studies were of no help. The eosinophil count rose to 41 per cent and on the tenth hospital day ascaris eggs were noted in the stool. He was treated with piperazine and improved rapidly. The edema subsided and the symptoms resolved; however, the lung infiltration persisted for 2 weeks. Eight months later the child was again seen with a mild upper respiratory infection of 1

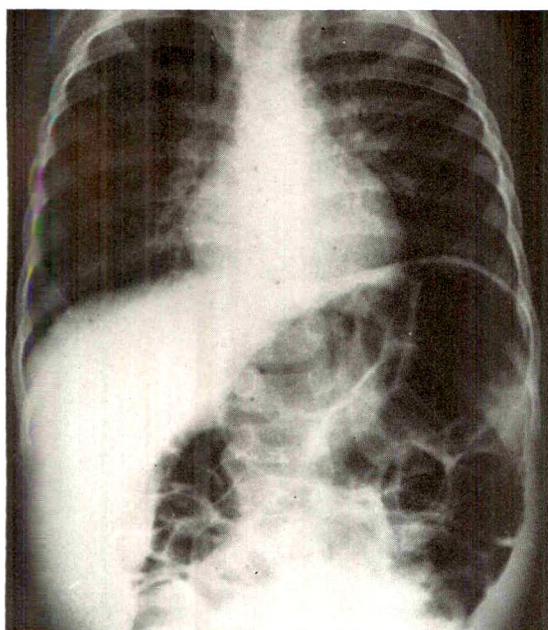


FIG. 3. Loeffler's pneumonia, small bowel distention and curvilinear outlines of ascaris in the left lower quadrant are seen—a case of reinfection.

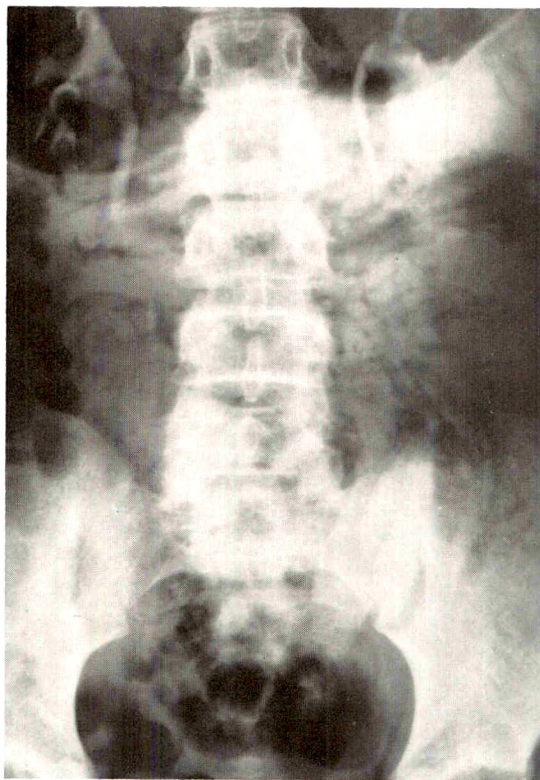


FIG. 4. An 11 year old child with edema of one foot showing massive ascaris infestation on this intravenous pyelogram. Multiple linear opacities are seen throughout the small bowel. The only symptom referable to the parasitism was restlessness in her sleep.

week's duration, cough, coryza, pitting edema of hands and feet, and "hives" of 1 day's duration. His temperature was 103° F. The physical examination revealed bilateral basilar pneumonia. Roentgenograms (Fig. 5) demonstrated pneumonia and ascaris in the abdomen. The stool was positive for ascaris, and he had a white blood cell count of 22,000 with 7 per cent eosinophils. His response to treatment with antihistamines and piperazine was again rapid. It is likely that this patient represents an allergic reaction to ascaris as suggested by Mercer *et al.*²⁴

Three patients who were followed from 3 to 9 months for peptic ulcer without clear recognition of an ulcer showed little change in symptoms. When the ascariasis was recognized on upper gastrointestinal roentgenograms in 2 patients and on plain roent-



FIG. 5. An erect posteroanterior chest roentgenogram demonstrates consolidating pneumonia in the right upper lobe, generalized bronchopneumonia, and ascaris in the left abdomen. The ascaris are seen as curvilinear opacities and are outlined by small bowel gas.

genograms in the third, proper treatment was instituted with lasting results.

In 5 patients surgery had been per-



FIG. 6. A 4 year old child with megacolon was incidentally found to have ascariasis during a barium enema examination. The worms are seen in axial projection outlined by gas in the small bowel between loops of barium filled colon. They appear as many rounded and ovoid opacities.

formed. One patient had an intestinal obstruction due to intussusception, 1 had appendicitis and 3 had only ascariasis.

A high incidence of dilated loops of small bowel, without obstruction, was seen in cases with abdominal symptoms. This is felt to be due to toxins produced by the worms causing irritability and spasm of the bowel.³² Conservative treatment resulted in rapid improvement of the patients and resolution of the signs and symptoms of intestinal obstruction. No deaths were encountered as a result of ascariasis in this series.

DISCUSSION

The presence of *Ascaris lumbricoides* is often overlooked on roentgenograms of the chest and abdomen in children unless the infestation is massive. The outline of these worms by gas in the small bowel makes recognition relatively easy, be there only one or many worms present. An increased index of suspicion and understanding of the varied roentgenographic appearance of ascariasis will greatly improve the incidence



FIG. 7. A supine roentgenogram of the abdomen demonstrates pica on the right and ascaris worms on the left. The association of pica and ascariasis is a common one.

of early diagnosis, especially when the symptoms are bizarre.

The roentgenographic signs of ascariasis can be divided into primary and secondary. The primary signs are diagnostic and consist of identification of single or multiple worms outlined by bowel air, either in linear masses (Fig. 4 and 5) or whirlpools,³³ or in axial projection (Fig. 6 and 8). A single worm can be more easily seen in a linear projection but a well-defined round opacity of a single worm in the axial ("on end") projection^{26,28} can often be diagnostic. The secondary signs are not diagnostic but the presence of two or more in a patient should be highly suspicious. They include the demonstration of pneumonia, pica (Fig. 7), ileus, lead lines,¹⁴ and simulated small bowel obstruction (Fig. 9).

It is noteworthy that the secondary signs are of less significance in teen age children and of little or no help in adults. This group of patients, however, forms a very small proportion of those with ascariasis.

The close correlation in these patients of

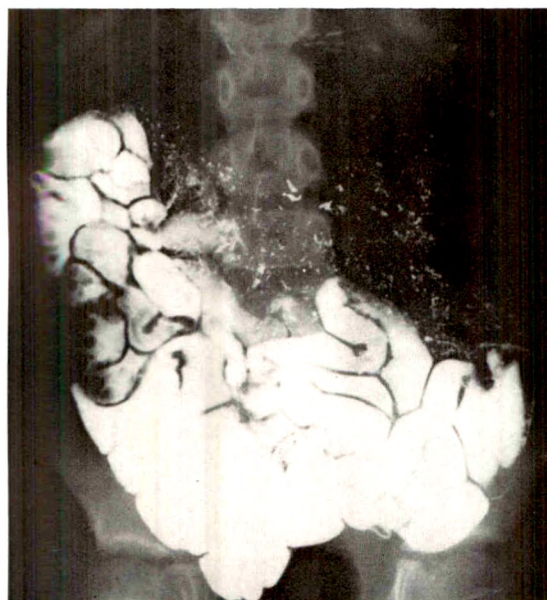


FIG. 8. A small bowel barium study demonstrates many ascaris worms in the right side of the abdomen. They are seen in axial projection and look like many round filling defects in the barium column. This illustrates why they are frequently seen only in the axial projection.

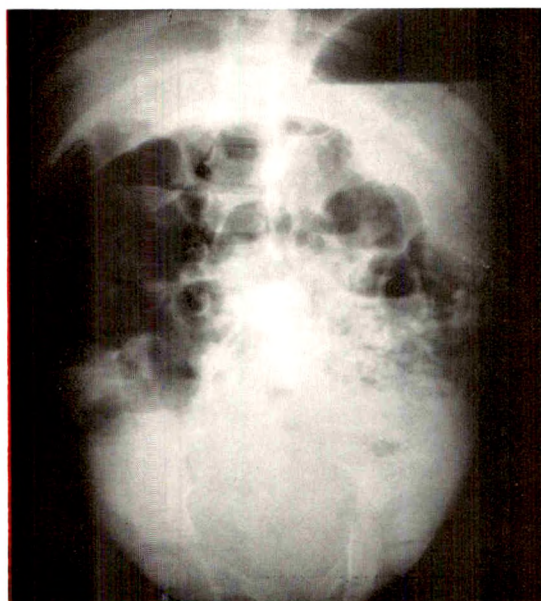


FIG. 9. A dilated small bowel with air fluid levels is evident in this erect study of the abdomen. Multiple ascaris worms are visualized in the left abdomen in both longitudinal and axial projections. The simulated small bowel obstruction seen here is a common occurrence and is probably due to toxins from the ascaris worms causing irritability and spasm of the bowel.

eosinophilia of a significant degree (greater than 10 per cent) with Loeffler's pneumonia is to be expected. The 2 cases in our group which did not fall into this expected pattern can be explained by the delayed appearance of eosinophilia as described by Le Melletier *et al.*¹⁸

SUMMARY

1. A review of 95 cases of ascariasis from Charity Hospital is presented and the clinical findings as related to the roentgenographic findings are described.
2. The ease with which the ascariasis can be seen on plain roentgenograms of the chest or abdomen is emphasized.
3. Typical roentgenograms showing the signs of ascariasis are presented.
4. Primary and secondary roentgenographic signs of ascariasis are discussed.

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METASTATIC CARCINOMA OF THE SMALL BOWEL*

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METASTATIC carcinoma of the small bowel and its mesentery, although a common finding at necropsy, may go unrecognized roentgenologically.^{1,2,3,5} A study of these patients reveals a multiplicity of roentgen changes which, while not pathognomonic, are highly suggestive. In some of these patients, the presence of abdominal carcinomatosis is obvious clinically and small bowel examination demonstrates findings indicative of multiple large metastatic masses. Difficulties, however, arise when a primary site is not known and the disease is less advanced. In such instances, only a single lesion may be discovered or there may be no constant morphologic alterations in the small bowel but only functional motor changes or ascites.

The most frequent sites of origin of metastatic disease to the small bowel are the ovary, pancreas, stomach, colon, breast, lungs and uterus. Patients usually present with symptoms indicative of intestinal obstruction, such as pain and increasing abdominal girth or with weight loss. Gastrointestinal bleeding may, occasionally, be the first manifestation of metastatic disease. Abdominal examination will usually reveal distention, intra-abdominal masses or ascites.

Pathologically, the metastatic lesion may originate in the wall of the bowel, subserosally, or in the mesentery. A submucosal lesion may develop a pseudo-pedicle and present as an intestinal polyp and cause intussusception and bleeding. More commonly, mesenteric or serosal deposits extend secondarily into the wall of the bowel, producing angulation, fixation and flattening of the contour of the bowel wall. With continued growth, the tumor may extend through the bowel wall and present as an intraluminal sessile mass with or without

ulceration. In other instances, the appearance of an intraluminal mass may be simulated by invagination of the wall and tumor toward the lumen. Intestinal obstruction is caused by a combination of encroachment on the lumen, local fixation and deformity, as well as angulation and twisting of the bowel.

For descriptive purposes, these metastatic lesions related to the small intestine may be arbitrarily divided into three groups depending on the predominating type of roentgen change. There is obviously considerable overlap of these groups.

CLASSIFICATION

Group I. Single or multiple metastatic lesions involving primarily the wall of the small bowel.

Group II. Metastatic lesions involving primarily the mesentery, with or without involvement of the adjacent wall of the bowel.

Group III. Multiple metastatic nodules involving both the small bowel and the mesentery. Ascites is particularly common in this group.

GROUP I

The earliest finding in Group I is that of a discrete intramural, extramucosal tumor which produces a contour defect similar to any other submucosal tumor (Fig. 1, A and B). Metastatic lesions usually show some irregularity or lobulation and often superficial ulcerations. With continued growth, the lesion may involve almost the entire circumference of the bowel wall and produce rather marked and irregular narrowing of the lumen and partial obstruction (Fig. 2). However, most of the growth may occur intraluminally with the production of a false pedicle and an intraluminal polypoid

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FIG. 1. (A) Metastatic carcinoma from body of uterus. There is a localized, 3 cm., slightly irregular submucosal nodule in the fourth portion of the duodenum (arrows). There is no mucosal ulceration or proximal dilatation. The defect does not have the exquisite sharpness usually associated with benign intramural tumors. This type of nodular defect can be seen also as a result of direct extension from a carcinoma of the body of the pancreas. (B) Metastatic carcinoma from the ovary. A solitary, submucosal lesion is present in the jejunum. The margins of the mass are not exquisitely sharp. Despite this, however, differentiation from a benign submucosal tumor is difficult.

defect simulating an adenomatous polyp (Fig. 3). This process may be associated with some degree of intussusception. However, long segments of intussusception are uncommon because of the frequent presence of fixation to the parietes. A metastatic lesion originating in the wall of the bowel may also, as it grows, extend into the mesentery and present as a so-called "endo-exo-enteric mass." When necrosis of the tumor is extensive, irregular ulceration similar to a primary carcinoma may be seen (Fig. 4, A, B and C). In an occasional case, there may be marked excavation of the tumor producing a huge irregular cavity within it.⁴

GROUP II

In Group II, the metastatic mass arises in the mesentery rather than in the wall of the

bowel. In the initial stage, single or multiple areas of smooth indentation upon the loops of small bowel are seen (Fig. 5A). At this stage, the small bowel loops are normally distensible. The appearance is not pathognomonic of metastatic disease since it is also seen with other retroperitoneal masses. When the mass infiltrates the bowel wall, the indentation may become irregular and fixation of the segment of bowel occurs associated with obvious evidence of extrinsic pressure along the mesenteric border (Fig. 5B). The involved part of the bowel is then flattened or convex towards the lumen. The folds in the area are flattened, angulated and blunted. As the mass continues to grow, the appearance is that of segments of small bowel wrapped around a mesenteric mass without evidence of mucosal ulceration or intraluminal poly-

polypoid masses (Fig. 5C). Obstruction in this group is usually associated with angulation and fixation of the intestinal loops. This is in contrast to lymphoma where compression of the bowel loop occurs but usually without fixation or angulation, or intimate adherence to the bowel wall.

GROUP III

In Group III, multiple implants produce changes not only in the wall of the bowel but also in the mesentery. This results in multiple areas of eccentric or concentric constriction with intervening segments of dilatation. The folds in the narrowed segment are distorted and not destroyed (Fig. 6A). The metastatic process in the mesentery produces shortening, rigidity and



FIG. 2. Metastatic carcinoma from the colon. There is a short, annular, constricting lesion with an eccentric lumen and slight proximal dilatation. The margins terminate abruptly without overhanging edges. Slight nodularity of the superior wall of the narrowed segment is also evident. There is no extrinsic component.



FIG. 3. Metastatic carcinoma from the kidney. There is a single polypoid intraluminal mass in the proximal jejunum (arrows) associated with a slightly eccentric lumen and minimal intussusception. A single small metastatic nodule such as this is difficult to differentiate from a submucosal tumor or intraluminal polyp.

thickening of this structure. As a result of the shortening of the mesentery, the small intestine appears short. Shortening of the mesentery is also associated with a peculiar crowding of the valvulae conniventes which may be referred to as "pleating" or "shirring." This roentgen appearance is similar to that noted in small intestinal intubation (Fig. 6, B and C).

The small bowel at the ligament of Treitz presents a rather special problem because both metastatic lesions and extension into the bowel from carcinoma of the pancreas are frequent in this area (Fig. 7A). Since the bowel in the region of the ligament of Treitz may be obscured by the overlying stomach, a supine or lateral decubitus projection may provide better visualization (Fig. 7B). Often the first suspicion of the presence of a tumor in this area arises from the presence of dilatation of the proximal portion of the duodenum.

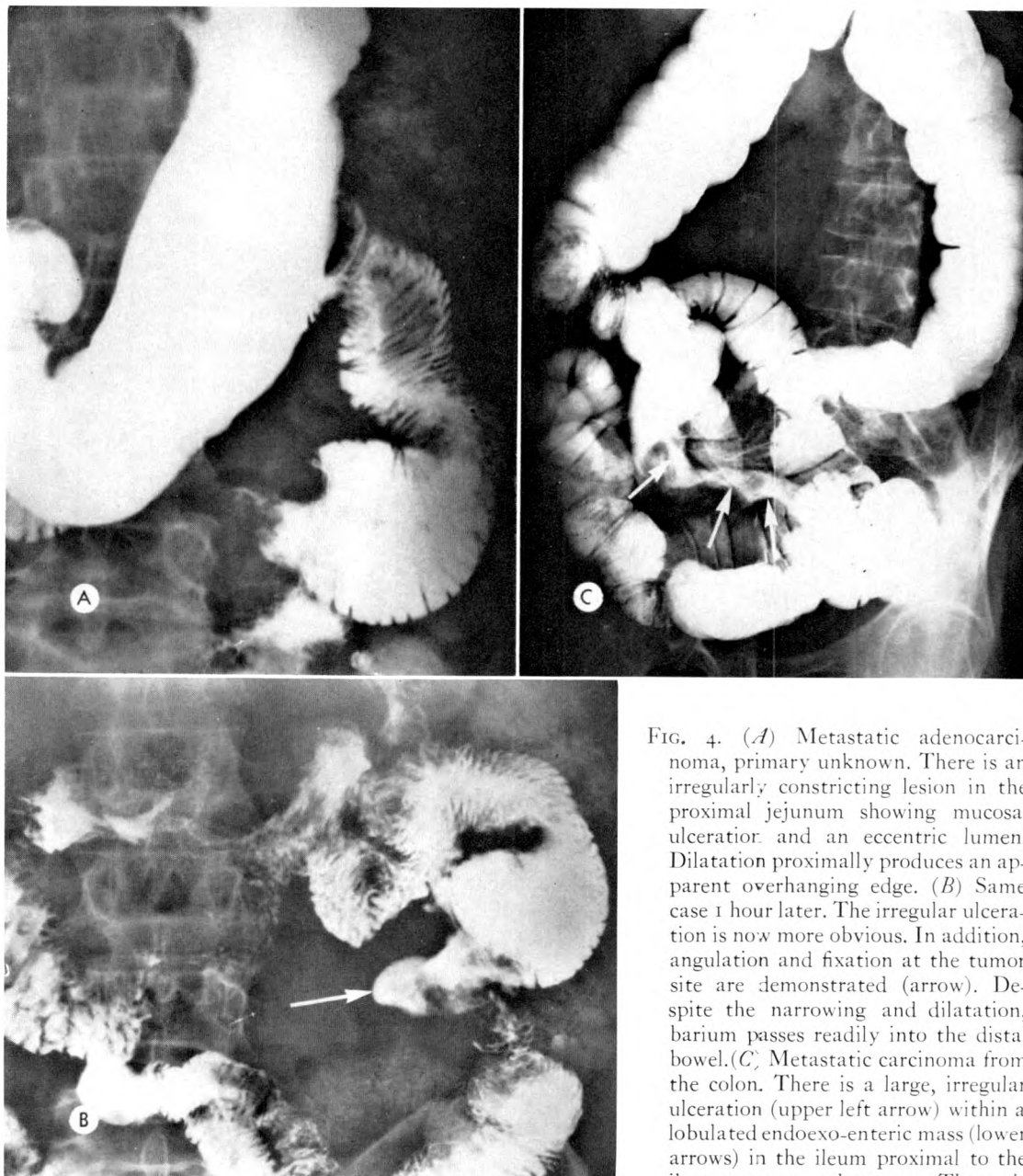


FIG. 4. (A) Metastatic adenocarcinoma, primary unknown. There is an irregularly constricting lesion in the proximal jejunum showing mucosal ulceration and an eccentric lumen. Dilatation proximally produces an apparent overhanging edge. (B) Same case 1 hour later. The irregular ulceration is now more obvious. In addition, angulation and fixation at the tumor site are demonstrated (arrow). Despite the narrowing and dilatation, barium passes readily into the distal bowel. (C) Metastatic carcinoma from the colon. There is a large, irregular ulceration (upper left arrow) within a lobulated endoexo-enteric mass (lower arrows) in the ileum proximal to the ileotransverse colostomy. There is

only minimal obstruction. This appearance is often seen with other submucosal tumors such as lymphosarcoma.

Distortion, fixation and stretching of the folds, irregular ulcerations and obstruction are features which should suggest an infiltrating neoplasm in the region of the ligament of Treitz.

As a result of extensive involvement of the mesentery and bowel wall, there may be

ascites. Ascites can be recognized by bulging flanks and the central position of the small intestinal loops. On occasion, a peculiar appearance of the valvulae conniventes, a "herring-bone" configuration, is seen (Fig. 8A). This pattern is presumably due to trapped air distributed throughout the

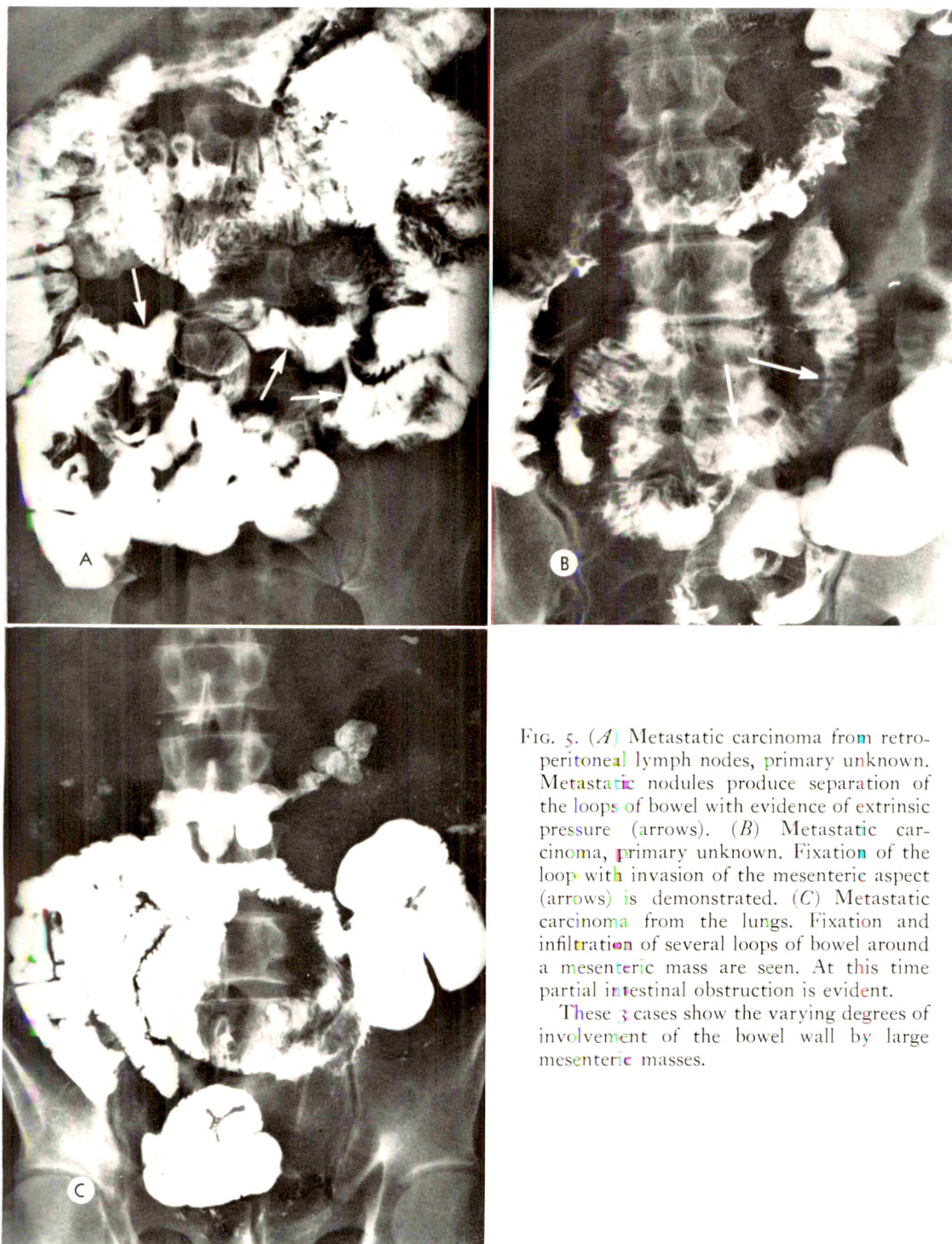


FIG. 5. (A) Metastatic carcinoma from retroperitoneal lymph nodes, primary unknown. Metastatic nodules produce separation of the loops of bowel with evidence of extrinsic pressure (arrows). (B) Metastatic carcinoma, primary unknown. Fixation of the loop with invasion of the mesenteric aspect (arrows) is demonstrated. (C) Metastatic carcinoma from the lungs. Fixation and infiltration of several loops of bowel around a mesenteric mass are seen. At this time partial intestinal obstruction is evident.

These 3 cases show the varying degrees of involvement of the bowel wall by large mesenteric masses.

bowel, producing an air contrast study. This is best seen with partial intestinal obstruction and is more commonly noted with metastatic lesions but not in ascites or

other etiologies. Because of the marked mesenteric involvement, there is fixation and drawing together of the bowel loops with central location of the loops which re-

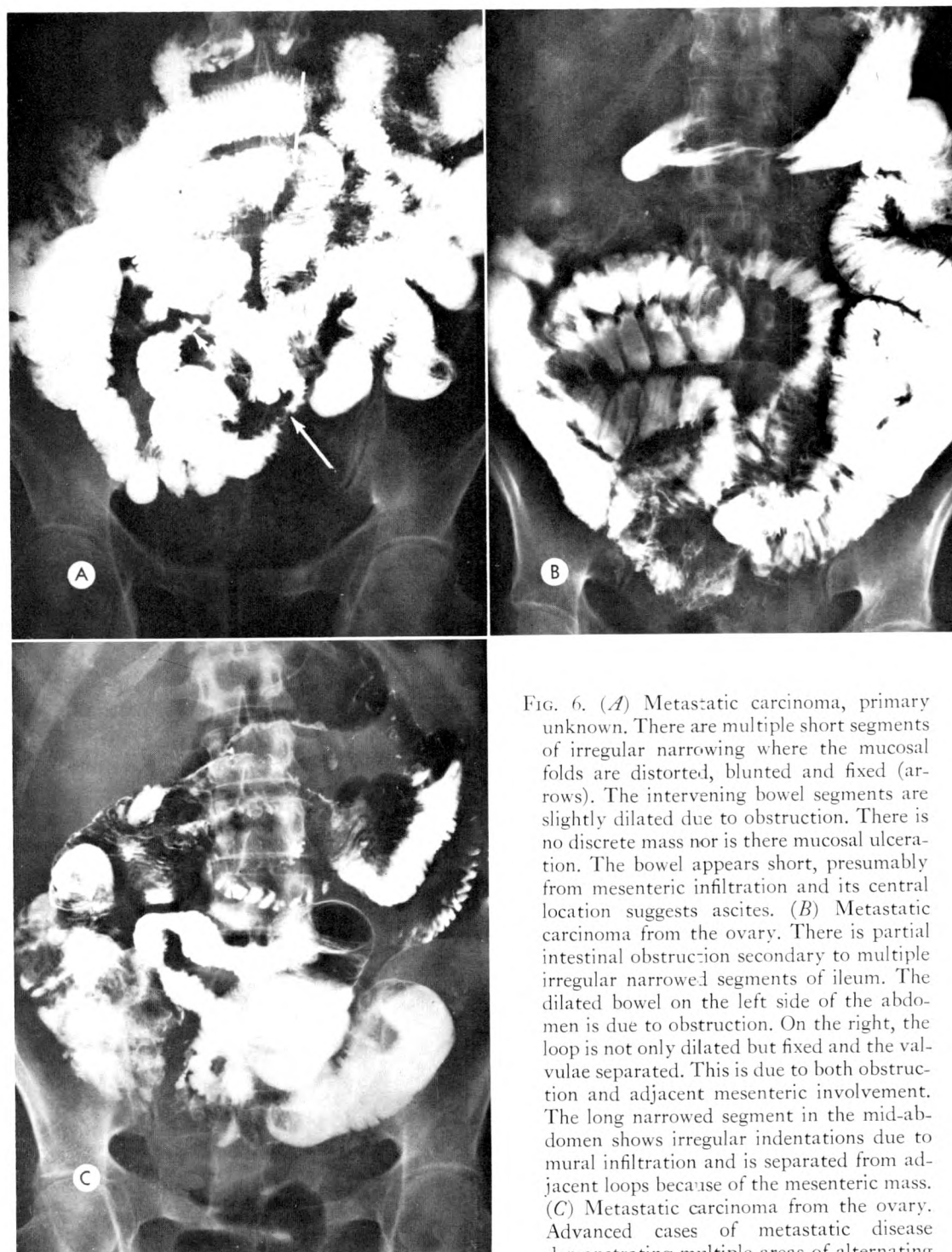


FIG. 6. (A) Metastatic carcinoma, primary unknown. There are multiple short segments of irregular narrowing where the mucosal folds are distorted, blunted and fixed (arrows). The intervening bowel segments are slightly dilated due to obstruction. There is no discrete mass nor is there mucosal ulceration. The bowel appears short, presumably from mesenteric infiltration and its central location suggests ascites. (B) Metastatic carcinoma from the ovary. There is partial intestinal obstruction secondary to multiple irregular narrowed segments of ileum. The dilated bowel on the left side of the abdomen is due to obstruction. On the right, the loop is not only dilated but fixed and the valvulae separated. This is due to both obstruction and adjacent mesenteric involvement. The long narrowed segment in the mid-abdomen shows irregular indentations due to mural infiltration and is separated from adjacent loops because of the mesenteric mass. (C) Metastatic carcinoma from the ovary. Advanced cases of metastatic disease demonstrating multiple areas of alternating narrowing and dilatation with fixation, angulation of the bowel loops, shortening and ascites.



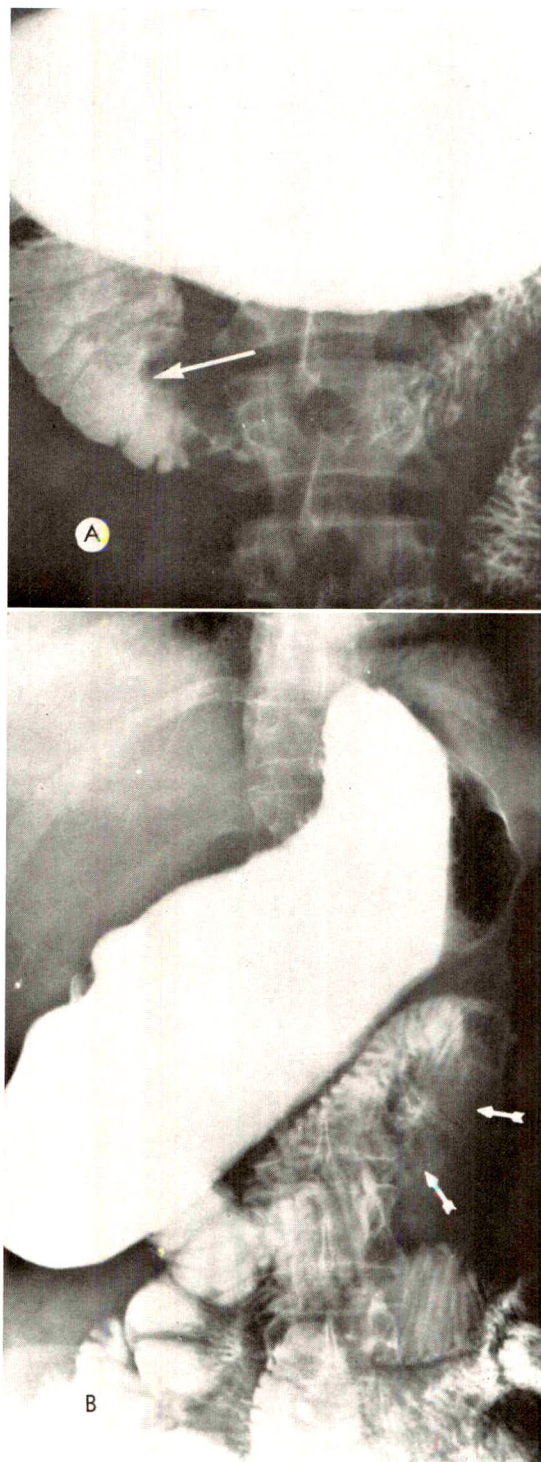
FIG. 7. (A) Metastatic carcinoma, primary unknown. Narrowed segment in the third portion of the duodenum (arrow) with thickened nodular folds and obstruction. It is impossible to distinguish invasion by carcinoma of the pancreas from metastatic implants in this region. (B) Metastatic carcinoma from the cervix. There is an obstructing lesion of the ligament of Treitz which is best visualized in the lateral decubitus position (arrows).

main fixed despite change in position of the patient (Fig. 8, B and C). Occasionally, the eccentric clumping and fixation of loops by carcinomatosis produce a pseudo-internal hernia configuration (Fig. 9A).

Plain roentgenograms of the abdomen may be diagnostic of carcinomatosis. These show segments of irregularly contoured, dilated loops of small bowel scattered in different portions of the abdomen. If this appearance is associated with bulging of the flanks and central location of the bowel, the presence of metastatic carcinoma is likely (Fig. 9B). Fixed soft intrusions on the gas column of dilated loops may also be seen. An occasional finding is the presence of multiple, small, psammoma-like calcifications. These are tiny, 1 to 2 mm. in diameter, and so faint that they may be overlooked (Fig. 9C). On occasion, the calcific densities in metastatic tumors may be large and irregular and superficially resemble calcified mesenteric lymph nodes or uterine fibroids. Metastatic calcifications, however, tend to be less confluent and dense in contrast to those seen in fibroids and lymph nodes where these are lumpy, confluent and sharply defined. Peritonitis with ascites due to an inflammatory process such as tuberculosis may produce some of the findings described above.

DIFFERENTIAL DIAGNOSIS

When a metastatic lesion presents as a solitary defect, differentiation from a primary tumor may not be possible. However, the classic appearance of a primary carcinoma with a short segment of stenosis and



symmetrical overhanging edges is uncommon with a metastatic process. The ulcerations in metastatic tumors, when present, are usually larger and more irregular, and

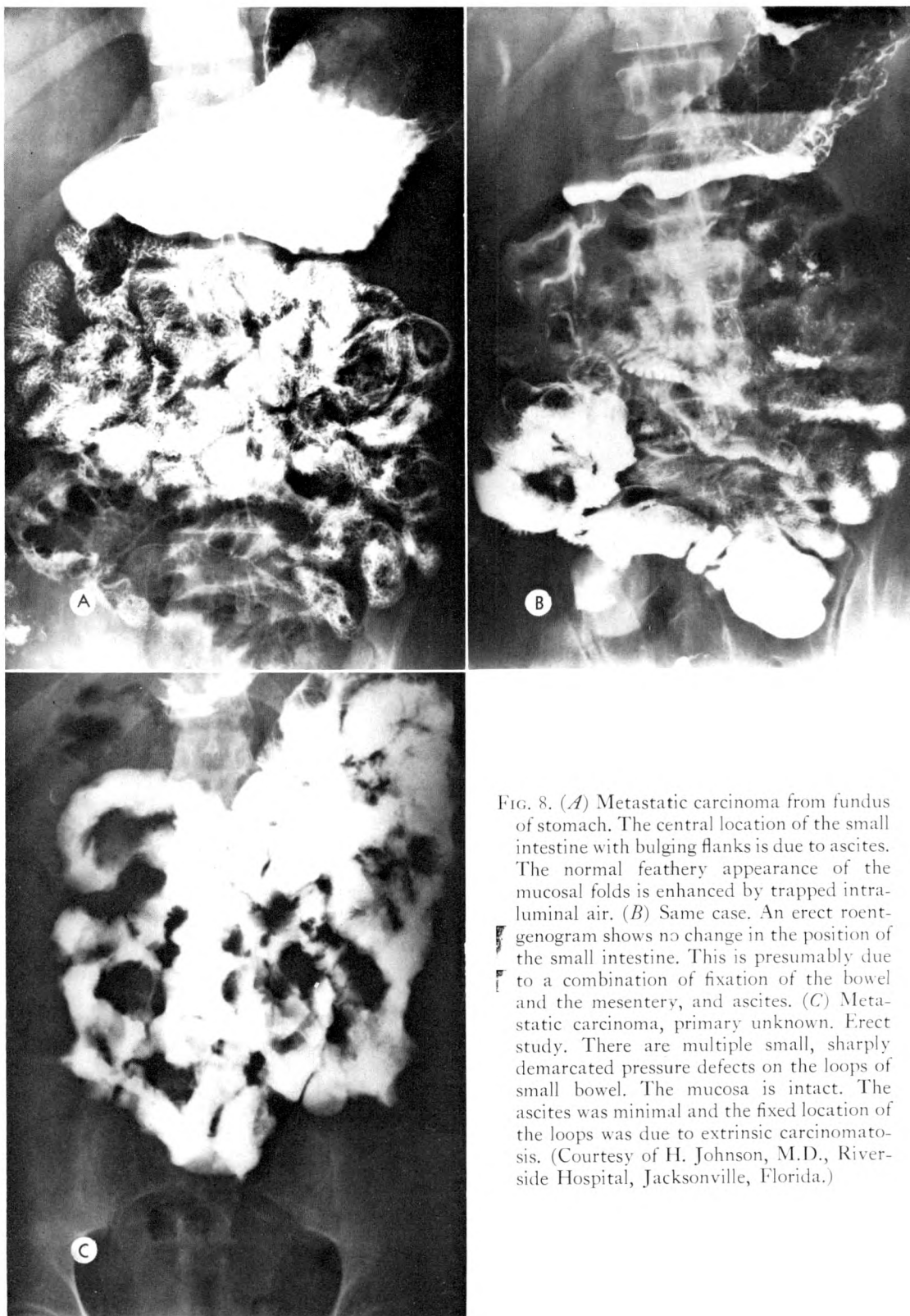


FIG. 8. (A) Metastatic carcinoma from fundus of stomach. The central location of the small intestine with bulging flanks is due to ascites. The normal feathery appearance of the mucosal folds is enhanced by trapped intraluminal air. (B) Same case. An erect roentgenogram shows no change in the position of the small intestine. This is presumably due to a combination of fixation of the bowel and the mesentery, and ascites. (C) Metastatic carcinoma, primary unknown. Erect study. There are multiple small, sharply demarcated pressure defects on the loops of small bowel. The mucosa is intact. The ascites was minimal and the fixed location of the loops was due to extrinsic carcinomatosis. (Courtesy of H. Johnson, M.D., Riverside Hospital, Jacksonville, Florida.)

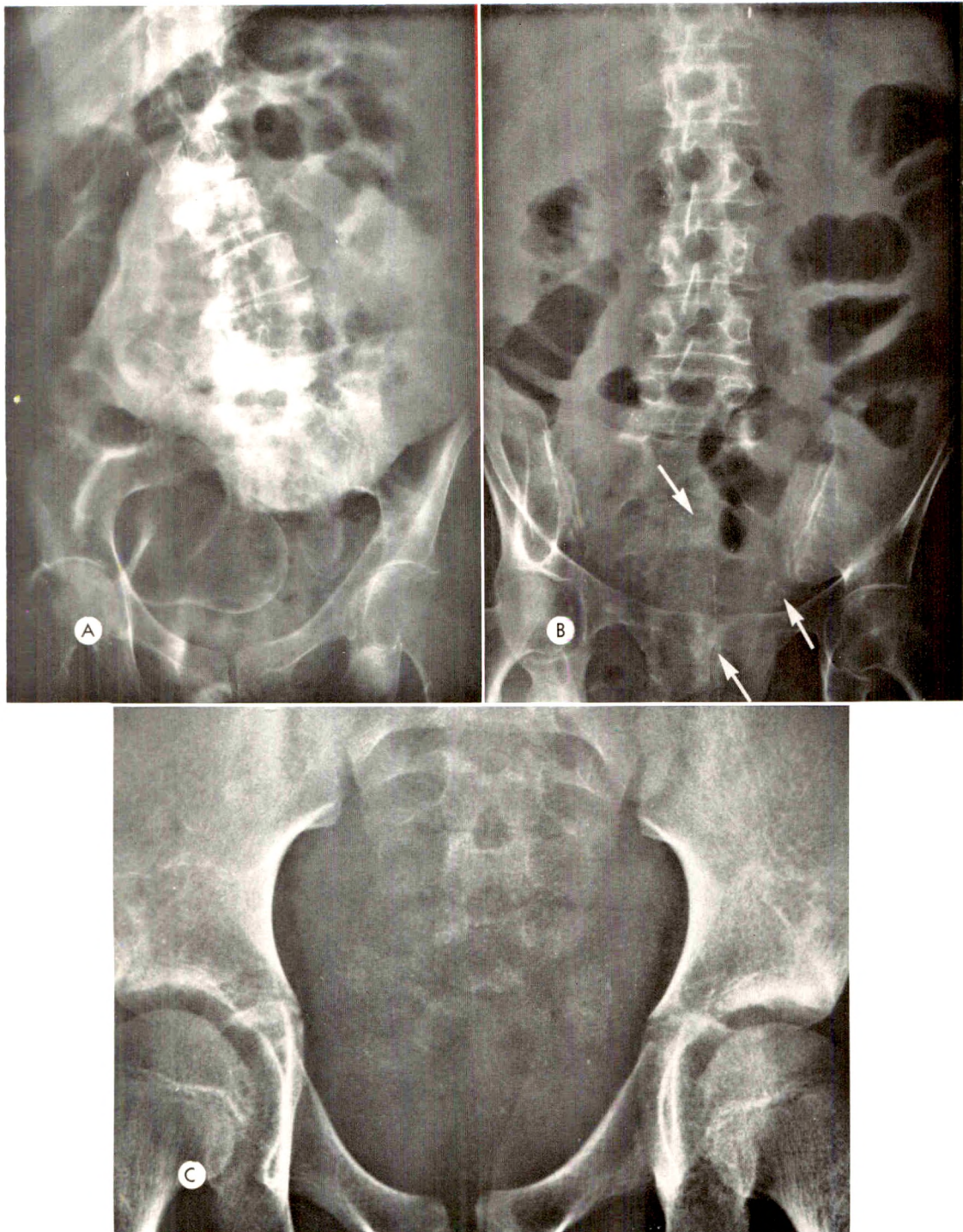


FIG. 9. (A) Abdominal carcinomatosis, primary unknown. The large central irregular mass is due to matted, fixed mesentery and small bowel infiltrated by metastatic carcinoma. Free intraperitoneal air outlines the cecum and accentuates the mass which simulates an internal hernia. (B) Metastatic carcinoma from the appendix. Multiple dilated small bowel segments, some of which have irregular contours and a conical appearance, are scattered in the abdomen. This is due to trapped air secondary to multiple partial obstructions. The calcifications are within metastases and are larger than psammoma bodies (arrows). (C) Multiple punctate psammoma-like calcifications are seen in the pelvis in a 11 year old child. Diagnosis: Undifferentiated carcinoma, probably arising in teratoma of ovary.

the bowel is frequently angulated and fixed at the site of the tumor. Intestinal obstruction, although present in both, is usually more marked in primary carcinoma. A metastatic process presenting as a single constricting lesion must also be distinguished from inflammatory lesions, a stricture secondary to infarction, and a lymphoma, specifically Hodgkin's disease. Irregularity of the contour and eccentricity of the process are features not ordinarily found in segmental infarction where the walls are more often tapering and conical. In regional enteritis, single short segments of involvement are rare and the inflammatory nature of the process will usually be evident by characteristic mucosal changes. In lymphosarcoma, there is often local dilatation of the bowel lumen despite the infiltrating nature of the tumor process. Hodgkin's disease, on the other hand, may produce narrowing and irregularity of the contour, as seen in metastatic or primary carcinoma. When a large mass is present, obviously displacing adjacent small bowel, both lymphoma and metastatic disease must be considered. Angulation and fixation as well as direct involvement of the bowel by retroperitoneal lymphosarcoma is not common and lymphoma of this type is rarely associated with any significant degree of obstruction.

The chief problem in differential diagnosis in the region of the ligament of Treitz is direct extension from a carcinoma of the pancreas. In most of these instances, the possibility of metastatic disease will arise only when a previous primary neoplasm has been evident.

Metastatic carcinoma which has caused shortening of the mesentery and multiple areas of narrowing and intervening dilatation may result in a superficial resemblance to granulomatous ileojejunitis. Absence of involvement of the terminal ileum suggests metastatic disease. Moreover, in metastatic carcinoma confined to the wall of the bowel or the adjacent mesentery, the mucosal

folds, although distorted and angulated, are still present, and diffuse ulceration and a cobble-stone pattern are not evident. Sinus tracts and fistulae are uncommon in metastatic carcinoma.

SUMMARY

Metastatic carcinoma of the small bowel produces a variety of roentgen findings which can be diagnostic of this entity. The roentgen features have been divided into three groups and depend on whether the bowel alone is involved, the mesentery, peritoneum or all three. When only the bowel is involved, the more common roentgen findings include contour or polypoid defects, ulceration, narrowing of the bowel lumen and partial intestinal obstruction. With involvement of the mesentery, fixation and angulation of the bowel occur. The conspicuous roentgen finding at this time is often shortening of the bowel and mesentery with shirring and pleating of the valvulae. When the peritoneum is involved, ascites may occur. An interesting roentgen configuration associated with the presence of ascites is a fixed pseudo-internal hernia appearance.

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HYDROCELE OF THE SPERMATIC CORD WITH ROENTGENOGRAPHIC FINDINGS SIMULATING A MUCOCELE OF THE APPENDIX

By ERNEST J. FERRIS, M.D.,* and IRVING A. SHAUFFER, M.D.†

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THE purpose of the authors is to describe a hydrocele of the spermatic cord, which was unique, both in its location and in its roentgenographic appearance. A brief discussion of the roentgenologic findings of this lesion with reference to its resemblance to a mucocele of the appendix is presented.

EMBRYOLOGY—ANATOMY

At the beginning of the third fetal month, sac-like pockets of peritoneum protrude into each side of the ventral abdominal wall. These are the beginnings of the vaginal processes. At the sixth fetal month, each sac evaginates through the ventral abdominal wall, thus producing a slanting inguinal canal.

The testicle, originating in its germinal analogue in the abdomen, migrates into the inguinal canal at approximately the seventh month. The ductus deferens, spermatic vessels and nerves are carried down into the scrotum along with the testis and epididymis. Before birth, the process vaginalis becomes pinched off from the peritoneal cavity. The sac, now isolated, becomes the tunica vaginalis. Its visceral layer is closely adherent about the epididymis and testis, whereas the parietal layer forms a lining into the scrotal sac.²

The internal inguinal ring is the cephalad limit of the spermatic cord and its parietal tunica vaginalis covering. The vas deferens courses out of the inguinal canal, proceeds retroperitoneally, and hooks around the ureter to enter the seminal vesical ejaculatory complex.⁵

PATHOLOGY

A hydrocele is an abnormal accumulation

of serous fluid in the sac of the tunica vaginalis.¹ While the majority are contained in the scrotum, there may be extension into the pelvis when the hydrocele involves the spermatic cord at or near the internal ring orifice.

Hydroceles may occur as a complication of gonorrhea, tuberculosis, or syphilis. Rarely, trauma or neoplasms are implicated in the etiology. They are usually asymptomatic. Acute symptoms may occur, due to secondary infection from the testicle or epididymis, or from twisting of its pedicle.³

ROENTGENOGRAPHIC FINDINGS

Hydroceles present as soft tissue masses, and are not specific. Soft tissue roentgenography of the scrotum will show a mass of water density. When the spermatic cord is the site of the hydrocele, soft tissue roentgenography is difficult because of overlying bony structures. On rare occasions, there may be some calcium in the wall of the hydrocele.¹

REPORT OF A CASE

T. P., Boston City Hospital #2004308. This 57 year old Negro male was in good health until the spring of 1962. He entered the Boston City Hospital with progressive dyspnea, hemoptysis, chills, and fever. Clinical and laboratory work-up, as well as a chest roentgenogram, were negative. At that time, he had bleeding hemorrhoids. A plain roentgenogram of the abdomen, made prior to a routine barium enema examination, showed a calcified mass approximately 6 cm. in diameter in the right lower quadrant. Both barium enema and intravenous pyelographic studies showed no abnormality. Clinical examination at that time was entirely negative. The presumptive diagnosis was a mucocele of the appendix. The patient refused surgery.

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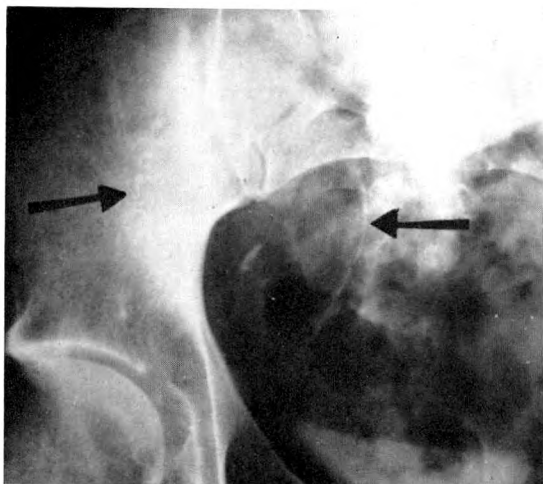


FIG. 1. Arrows denote a 6×8 cm. ovoid soft tissue density with a fine rim of calcium at its periphery. This extends upward from the right side of the pelvis and partially overlies the sacroiliac articulation.

The patient re-entered the Boston City Hospital, on March 3, 1964, with a chief complaint of recurrent bleeding hemorrhoids. Routine physical examination disclosed a hard mass about the size of a fist in the right lower quadrant.

Roentgen Findings. A preliminary barium enema study of the abdomen showed a soft tissue mass with an oval fine rim of calcium in the right lower quadrant (Fig. 1). The cecum appeared superiorly displaced (Fig. 2). There was incomplete filling of the appendix. With change of position, the calcified mass was not separated

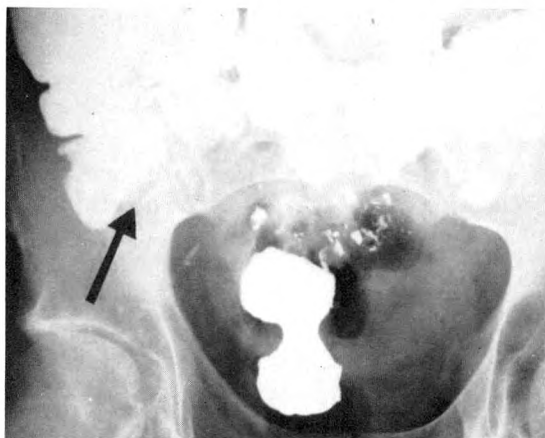


FIG. 2. Colon partially outlined with opaque material. The soft tissue mass in the right lower quadrant displaces the cecum superiorly with compression of its inferomedial aspect (arrow).

from the base of the cecum. On the roentgenogram made after evacuation, there appeared to be concentric compression of the mucosal folds at the base of the cecum, suggesting some degree of invagination (Fig. 3).

The apparent attachment of the mass to the base of the cecum, the calcific deposition in its wall, and the incompletely filled appendix led to the preoperative diagnosis of a mucocoele of the appendix.

Continuation of Hospital Course. On March 6, 1964, under spinal anesthesia, the abdomen was opened through a right paramedian incision. Immediately presenting in the right lower quadrant was a large 8 cm. cystic mass, retroperitoneal in location, displacing the cecum cephalad. Dissection showed that it extended down and into the inguinal canal (Fig. 4). The cyst contained thin serous fluid.

Pathology. The specimen measured 8×6×4.5 cm. Its wall measured 0.3 cm. in thickness. The inner lining of the cavity showed papillary projections which were identified as calcific in nature. Microscopic diagnosis was consistent with a hydrocele arising from the spermatic cord.

DISCUSSION

In view of the similarity of the roentgenographic findings in this case to those usually associated with a mucocoele of the appendix, it may be well to review the criteria for the diagnosis of mucocoeles of the appendix set forth by Euphrat.⁴ These are the following:



FIG. 3. Postevacuation roentgenogram demonstrates the constant relationship of the right lower quadrant mass to the inferomedial aspect of the cecum (arrow).

1. A sharply circumscribed lobular or reniform soft tissue mass with considerable mobility, but firmly attached to the cecum.

2. Medial displacement of the cecum by this mass.

3. Calcium deposits in the wall or substance of the mass, failure of the appendix to fill with contrast agent, and a vertical appearance of the fold of the cecal mucosa.

In spite of these criteria, the diagnosis of mucocele of the appendix without calcification is extremely difficult. It is of interest to compare the roentgenograms in the described hydrocele of the spermatic cord with a calcified mucocele of the appendix, which was encountered at about the same time (Fig. 5 and 6). The striking similarity between the presented case of hydrocele of the spermatic cord and the case of mucocele of the appendix is quite obvious, and this entity must be added to the differential diagnosis of a right lower quadrant mass.

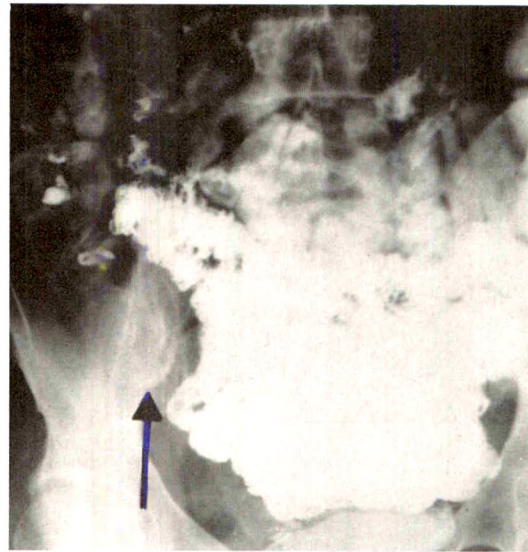


FIG. 5. Mucocele of the appendix. An ovoid soft tissue mass overlies the right sacroiliac articulation. A rim of calcium outlines its periphery (arrow). Bowel loops of the terminal ileum are displaced medially by this mass. The base of the cecum is compressed.



FIG. 4. Photograph at surgery. The hydrocele has been reflected downwards over the inguinal region. Arrow points to origin at internal inguinal ring.

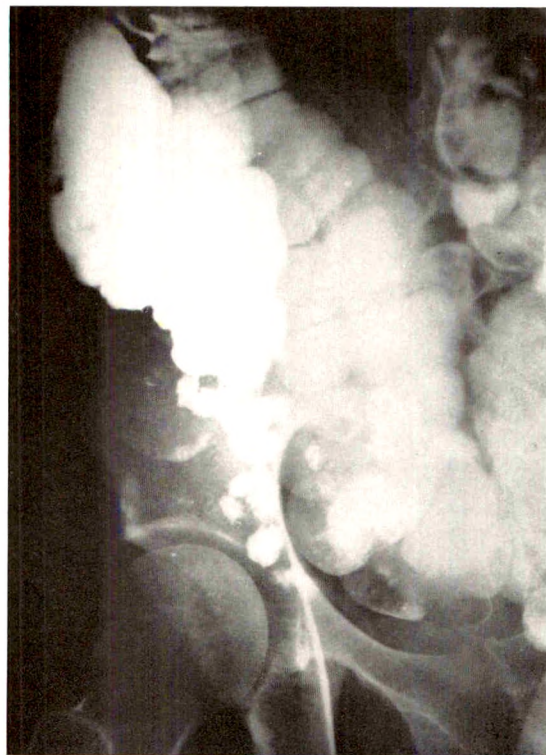


FIG. 6. Oblique roentgenogram of the right lower quadrant shows the relationship of the mucocele to the cecum.

SUMMARY

A case of a hydrocele of the spermatic cord is reported.

The roentgenograms showing a mass with a fine rim of calcification invaginating and adhering to the base of the cecum are presented.

The simulation of a mucocele of the appendix is stressed.

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DISPLACEMENT OF THE INTESTINE BY THE ILIOPSOAS MUSCLE*

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IT IS apparently not generally appreciated that the normal iliopsoas muscle may produce a remarkable displacement of the sigmoid colon or distal ileum, particularly in young muscular individuals. In some instances this may be confused with an abnormal pelvic mass displacing the bowel.

REPORT OF A CASE

A 29 year old female entered the University of Michigan Medical Center because of vaginal

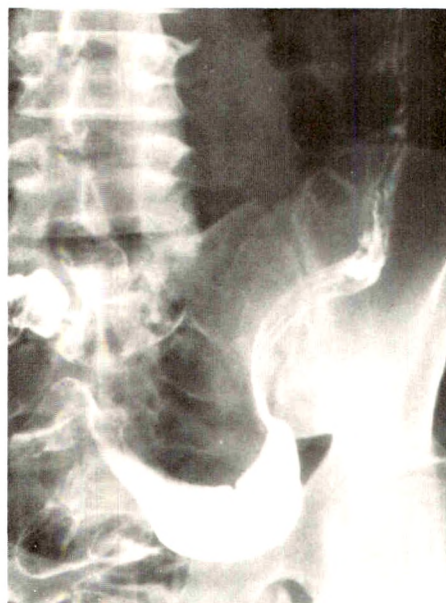


FIG. 2. Extrinsic pressure deformity of the sigmoid colon.

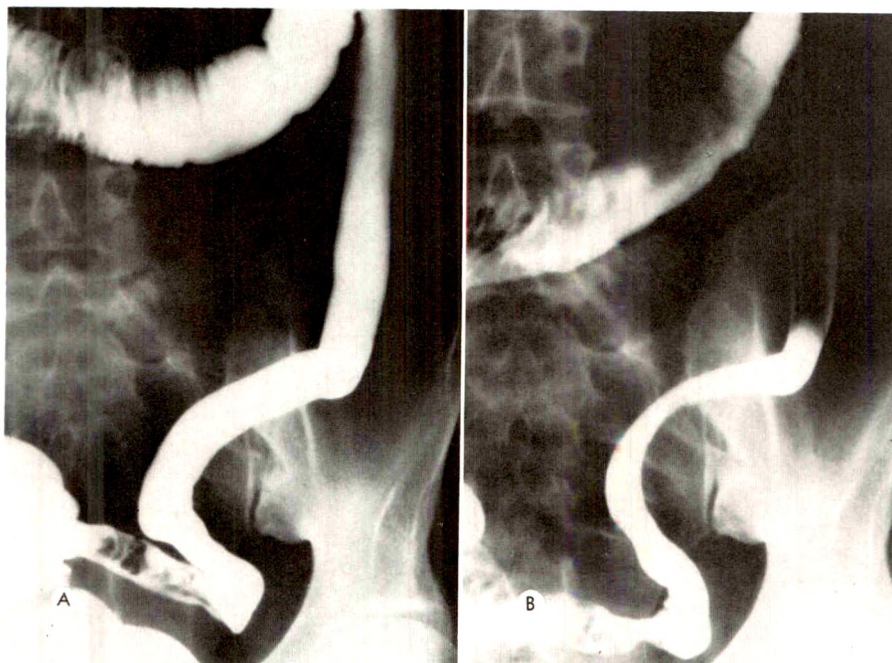


FIG. 1. (A) Pre-evacuation roentgenogram. (B) Post-evacuation roentgenogram. Anterior and upward displacement of the sigmoid colon near the left iliac fossa.

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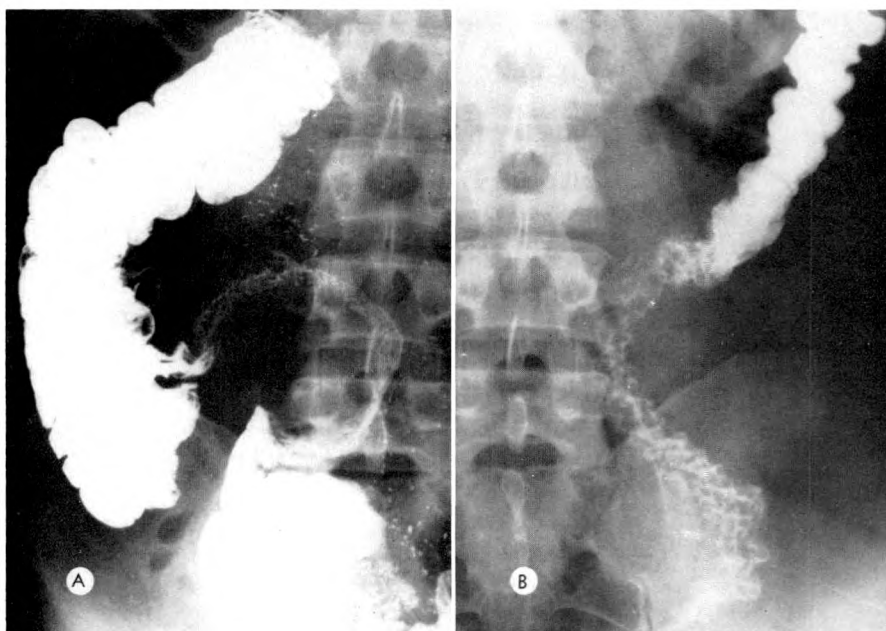


FIG. 3. (A) Small bowel examination showing extrinsic pressure deformity of the terminal ileum felt to be due to the psoas muscle. (B) Barium enema study showing symmetric displacement of the sigmoid colon at the same level.

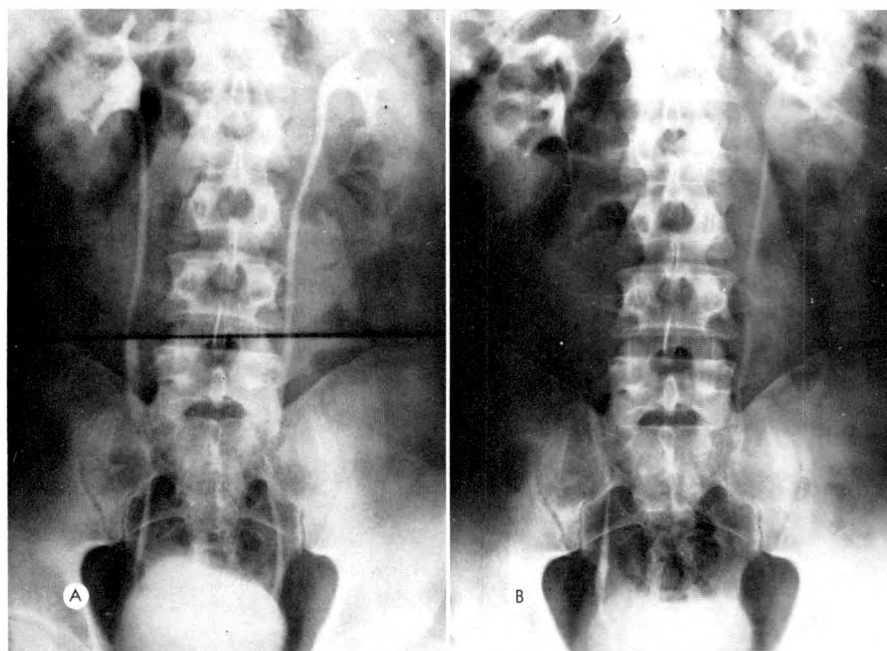


FIG. 4. Same case as in Figure 3, A and B. Intravenous pyelograms. (A) With compression. (B) Without compression. Both ureters veer medially and symmetrically as they cross the iliopectus muscles. Note the bilateral prominence of the iliopectus muscles.

bleeding of 2 months' duration. The patient was a well developed, white female in whom physical examination revealed the uterine cervix to be completely involved by an exophytic carcinoma which seemed to extend slightly toward the left fornix. Biopsy of the lesion showed undifferentiated squamous cell carcinoma. Barium enema examination demonstrated an arcuate, anterior and upward displacement of the sigmoid colon near the left iliac fossa (Fig. 1, *A* and *B*). Pelvic extension of the neoplasm with colon displacement was suspected. Intravenous urograms were normal but the lower ureters were not visualized. At celiotomy, it was found that the neoplasm was confined to the cervix uteri and adjacent vaginal wall. The left ovary and fallopian tube were normal. Both iliopsoas muscles were symmetrically large and it was felt that the sigmoid deviation was due to this factor.

COMMENT

Two other cases showing similar intestinal displacement have since been observed, although in neither was there surgi-

cal verification of this finding. In one elderly female patient, the sigmoid colon was displaced anteriorly and upward within the pelvis (Fig. 2). There was no clinical evidence of a pelvic mass and the barium enema examination was requested largely as a routine procedure. The second patient, a young muscular male, showed symmetric displacement of the terminal ileum and sigmoid colon, but this occurred above the pelvis at the level of L4-5 (Fig. 3, *A* and *B*). Re-examination showed that these segments of the bowel were not fixed in position and that there was no intrinsic abnormality of the intestine. Furthermore, intravenous pyelograms (Fig. 4, *A* and *B*), obtained to determine whether there was symmetric displacement of the ureters by the iliopsoas muscles, showed this to be the case.

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ROENTGEN SIGNS OF INTESTINAL NECROSIS*

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NECROSIS of the bowel is commonly associated with various forms of vascular occlusion. The latter may arise as a result of a simple mesenteric thrombosis either arterial or venous; the closure may be the result of vascular disease, or uncommonly from trauma, especially during surgery. Other forms of vascular occlusion are associated with a number of diseases of more obscure origin. It is notable that intestinal necrosis has been reported in patients with intact arteries and veins, apparently associated with digitalization.² Strangulation of the circulation of the bowel also occurs in connection with certain types of intestinal obstruction.

Intestinal necrosis may be the result of blunt trauma to the abdomen, possibly due to injury of the smaller mesenteric vessels. Finally, a necrotizing gastroenterocolitis from a virulent infection may accompany diabetes or the late stages of debilitating diseases. This has been observed especially in the lymphomata by Gildenhorn *et al.*,³ sometimes accompanied by abscess formation.

The determination of the presence of intestinal necrosis is of importance since it may be a good diagnostic sign of mesenteric thrombosis or of the other lesions which have been referred to above. Furthermore, the prognosis and management of the patient will be greatly influenced by the knowledge that necrosis is either impending or present.

ROENTGEN SIGNS

Roentgen signs may be crucial in establishing such a diagnosis and in this presentation we wish to delineate the essential features of intestinal necrosis which are manifested in simple roentgenograms of the

abdomen, without artificial contrast material.

The roentgenologic findings of vascular occlusion have been described on many occasions, but the evidences of necrosis secondary to such vascular occlusion have only been reported in more recent years. The first of these was a report by Wolfe and Evans,¹¹ in 1955, in which they indicated the presence of gas in the portal veins as a sign of some type of necrotic process occurring in the gastrointestinal tract. Later Susman and Senturia⁸ reported the same finding with a discussion of the source of the gas in the portal vein. They also discussed the differential diagnosis from gas in the biliary tract. Wiot and Felson⁹ reported a larger series of cases and in several cases they also called attention to the presence of gas in the intramural portion of the bowel demonstrated at autopsy. More recently, Barrett¹ has reported similar findings of gas in the portal circulation as a result of mesenteric occlusion.

In 1963 another sign of intestinal necrosis, that is, the presence of gas in the intramural portion of the bowel as distinct from pneumatosis intestinalis was described in detail by Schorr,⁷ who reported 2 cases and emphasized the importance of the roentgen signs as a diagnostic aid in cases of mesenteric occlusion.

The roentgen findings in mesenteric thrombosis or other vascular occlusions in the abdomen are variable and often difficult to interpret. In the early stages the most outstanding feature is a disproportion between the clinical findings, which are usually severe, and the minimal roentgen signs exhibited in simple roentgenograms of the abdomen without contrast. In fact, the presence of such minimal roentgen signs,

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with relatively little visible gas in the intestinal tract, in the face of a serious abdominal crisis, should always lead to consideration of vascular occlusion. The strangulation of the circulation of the bowel associated with intestinal obstruction gives a fairly striking picture which was described by Mellins and Rigler¹ in 1954. The important feature here is the presence of a solitary loop of bowel greatly distended with an excessive quantity of fluid within it. A similar feature can be found in mesenteric thrombosis without intestinal obstruction (Fig. 1). In this case there is also evidence of mucosal edema. As the process advances, a generalized distention of the bowel takes place. This has been described as being confined to the small bowel and the proximal colon up to the splenic flexure,

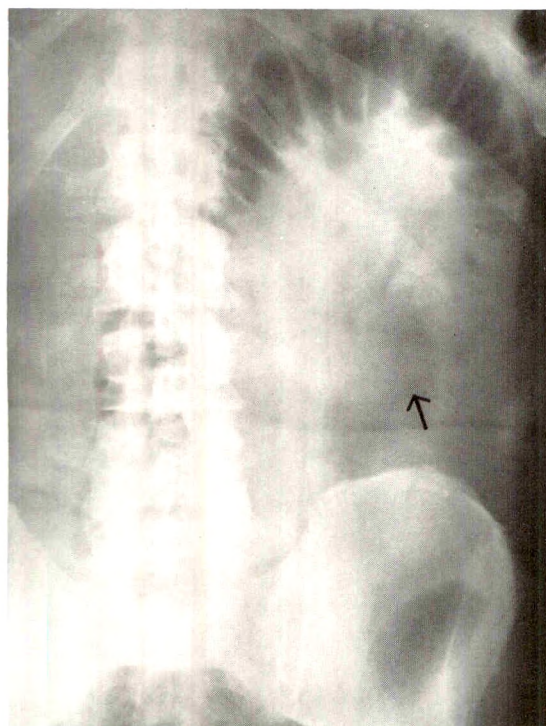


FIG. 1. Mesenteric thrombosis with eventual intestinal necrosis. First roentgenogram made 12 hours after onset shows isolated, gas distended loop of bowel frequently seen in early stages of mesenteric thrombosis or in strangulation obstruction. Arrow points to fluid filled loop with some edema of mucosa which is also an early sign of mesenteric thrombosis before necrosis.

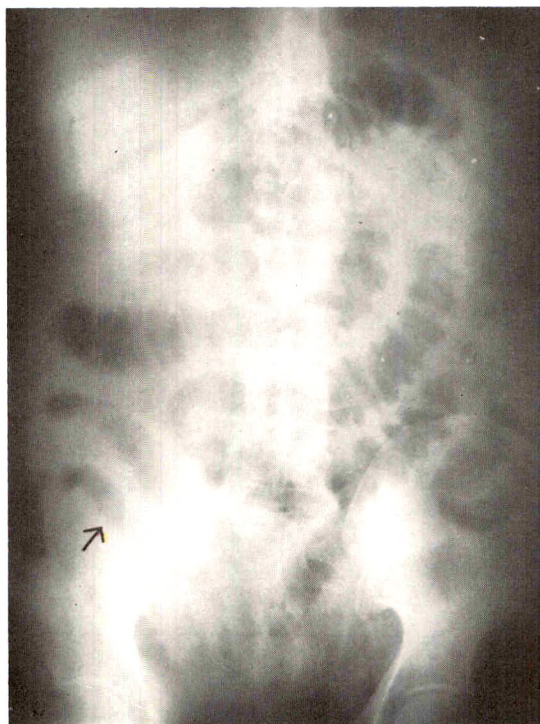


FIG. 2. Mesenteric thrombosis 36 hours after onset. There is fluid in the peritoneal cavity as shown by the separated gas filled loops of bowel. In the right lower quadrant, two small bowel segments are shown with narrowed lumina (arrow) and wide separation from each other. This combination indicates severe edema of the bowel wall, suggesting impending necrosis.

when it is the superior mesenteric artery which is occluded. Actually, it is uncommon to find this characteristic picture, but generalized distention of the small bowel is frequent with often some signs of colon distention as well. A more striking and more characteristic sign, and one which is indicative of impending intestinal necrosis, is the presence of several loops of bowel in which the lumen is greatly reduced but the wall is markedly thickened (Fig. 2). The latter can be determined by the separation of the remaining loops of bowel from those involved. The thickening of the bowel wall, of course, is due to the edema, congestion, and beginning gangrene which is present. This sign was first reported by Nelson and Eggleston² in 1960.

With the onset of true necrosis, in which

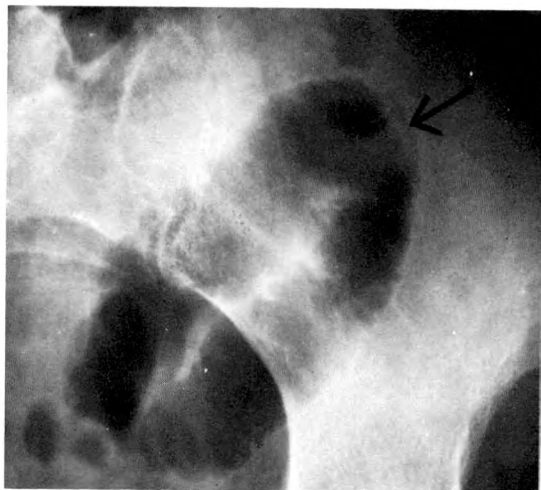


FIG. 3. Case of mesenteric thrombosis with intestinal necrosis. The semilunar, linear area of gas density in the wall of the bowel (arrow) is well shown. This is characteristic of the sign reported by Schorr, indicating extension of gas from the lumen of the bowel into its wall.

the mucous membrane is often destroyed sufficiently so that a communication between the lumen of the bowel and the intramural tissues is established, a layer of gas will appear in the subserosal or intramuscular areas of the bowel wall, as first reported by Schorr.⁷ He described this as a crescentic, linear shadow, the roentgenographic density of which is that of gas, outside the lumen of the bowel, and exhibiting between itself and the gas filled lumen a characteristic strip of increased density representing the bowel wall itself (Fig. 3). Occasionally, these shadows are "ring-like," surrounding the bowel entirely or semicircular in character (Fig. 4). It is important to distinguish these lines from the numerous radiable areas of little significance such as those caused by fat layers between the loops of bowel, beneath the liver, and those which occur as a result of the superimposition of one distended loop upon another. These can be distinguished because they are not confined to the bowel wall and are usually fairly straight rather than crescentic. They must also be differentiated from the appearance of pneumoperitoneum, seen in the horizontal position, in which both the inner

and outer walls of the bowel are visible.⁶ The gas in the peritoneal cavity is distributed fairly evenly between the bowel loops throughout the abdomen (Fig. 5).

Another possible source of error is a result of the presence of feces and gas poorly mixed within the bowel. Under these circumstances, there may be a ring-like shadow of translucency around an intraluminal mass. Usually, however, this can be distinguished by the marked irregularity of the mass and by the fact that the bowel wall is clearly seen outside the semicircular ring as an area of greater density.

Intestinal intramural gas from necrosis (Fig. 3, and 4) differs sharply from that found in pneumatosis intestinalis, which is probably of very different origin. In the latter, the patient is rarely seriously ill in contrast to patients with intestinal necrosis. The roentgen shadows are cystic or bubble-like and rarely of the linear character described above. The two terms have been used interchangeably, that is, pneu-

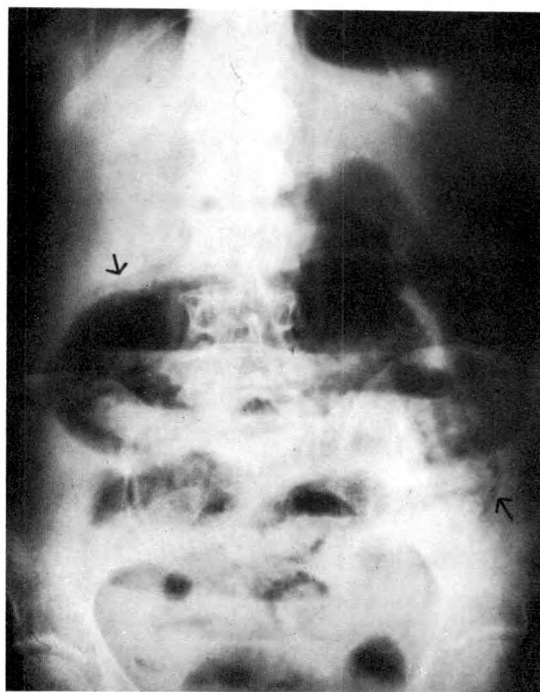


FIG. 4. Mesenteric thrombosis with intestinal necrosis. Multiple linear shadows of gas in the wall of the bowel (arrows) are well shown along with distended loops of bowel.

matosis intestinalis and intramural gas formation, but we believe they should be sharply distinguished.

Wolf and Marshak¹⁰ have described a long, linear area of lesser density appearing to be in the wall of the colon and occurring in patients with toxic megacolon secondary to ulcerative colitis. We have seen several such cases. The appearance resembles somewhat the presence of intramural gas as seen in intestinal necrosis but differs in important particulars. It is much longer, confined to the colon, and greater in density than intramural gas. We are not certain of the origin of this shadow, although Wolf and Marshak believe it is a fat line.

It is possible to demonstrate the breaks in the mucosa of the intestine if absorbable contrast medium is given by mouth and follow-up roentgen examinations are made. Usually, this is difficult to do since the patient is seriously ill, but in a case, studied

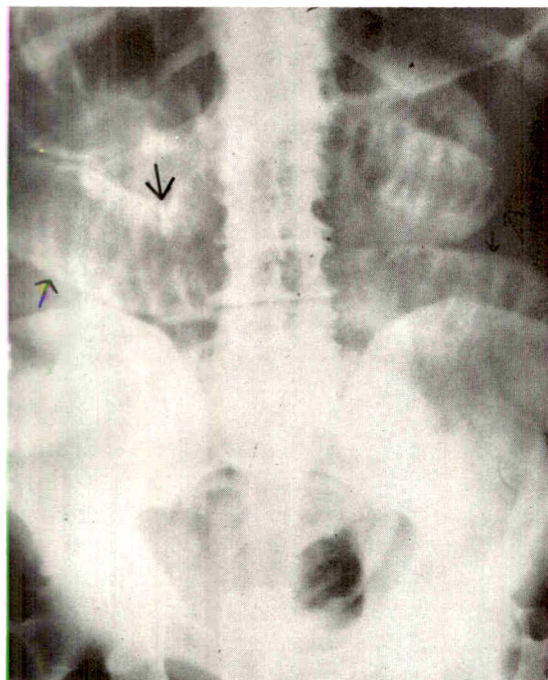


FIG. 5. Pneumoperitoneum from ruptured ulcer demonstrated in supine position. Note the visibility of both inner and outer walls of the bowel (arrows). There is no gas shadow because the gas is so uniformly and widely distributed and not confined within the bowel wall.

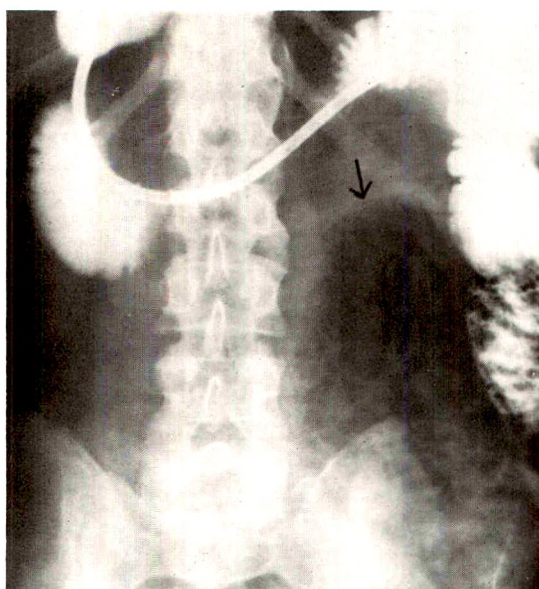


FIG. 6. Case 1. The segment of necrotic bowel is indicated by arrow. The linear and irregular areas of gas in the wall of the bowel seen here were present on a roentgenogram made 24 hours earlier. Just prior to this study, an iodine compound had been given by mouth after the introduction of a suction tube and the contrast medium is shown extending into the small bowel just proximal to the area of necrosis.

by Dr. Stanely Lance, exactly this situation pertained (Fig. 6, and 7).

REPORT OF A CASE

CASE 1. A male aged 56 years, was admitted on April 13, 1964 to the Huntington Memorial Hospital in Pasadena, California, complaining of a sudden attack of severe periumbilical pain 10 hours before admission, followed by a bout of diarrhea, nausea, vomiting, and accompanied by continuous pain. The first roentgenogram made on April 13, 1964 showed a solitary loop of bowel, markedly distended, with a large amount of fluid within it. Just beneath it another loop showed mucosal edema. It was thought to represent a "sentinel" loop, reflecting a nonspecific ileus, possibly the result of an acute abdominal condition. Exploratory laparotomy was done, an umbilical hernia was repaired, but no other evidence of abnormality was found. The mesenteric vessels were not completely explored but the small bowel was carefully inspected.

The patient remained toxic, developed a

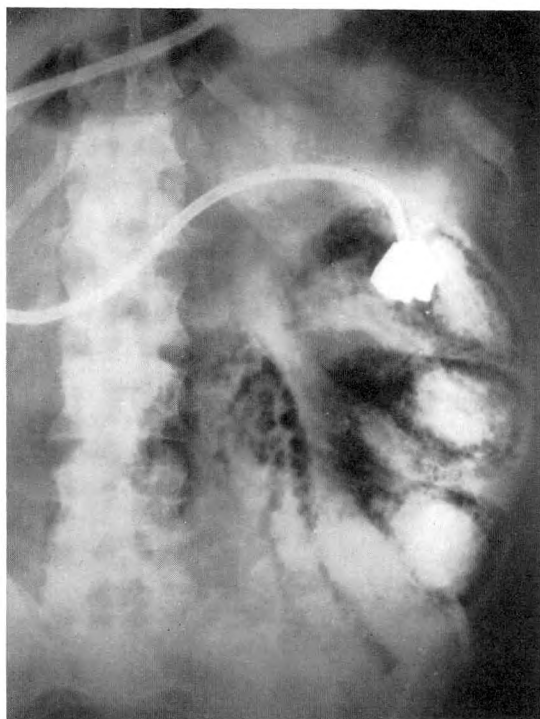


FIG. 7. Case 1. This study followed Figure 6 by approximately 30 minutes and the contrast material has now entered directly into the portion of bowel which is necrotic. The intramural layers of gas lying between the lumina of the loops of bowel are more clearly delineated because of the greater contrast produced by the iodine compound. Absorption through the wall of the bowel into the circulation occurred very rapidly so that in 30 minutes the urinary bladder was opacified and was demonstrated in roentgenograms.

minor degree of pneumonia, which cleared up, and then began to show evidences of obstruction. On April 21 a roentgenogram exhibited distended loops of bowel surrounded by irregular areas of gas, obviously outside the lumen of the bowel, but within the bowel wall. The condition of the patient, however, did not seem to correspond with so serious a situation as intestinal necrosis so that on the following day an organic iodine compound was given by mouth (Fig. 6). The same findings of gas in the wall of the bowel were shown, the contrast medium not yet in the necrotic loop. Thirty minutes later (Fig. 7) the contrast medium had advanced into the necrotic loops. The contrast material filling the lumen of the bowel accentuated the shadows of the gas in the walls of the intestine which were demonstrated as ir-

regular linear areas of gas density. Within 30 minutes, the urinary bladder became moderately opacified. This seemed to indicate absorption through the walls of the bowel at a much more rapid rate than could possibly occur in the normal. A diagnosis of infarction of the bowel was, therefore, made and the patient was again explored. At this time the small bowel was found to be necrotic, extending from about 3 inches distal to the ligament of Treitz to the mid-portion of the ileum. A small segment of the ileum and the entire colon appeared to be viable. The mesenteric artery was found to be thrombosed. Extensive resection of the small intestine was accomplished but the patient went downhill and died 2 days later. The specimen removed was reported by the pathologist to show gangrene of portions of the small intestine with extensive ulceration and inflammation. In some areas there was almost complete necrosis of the bowel wall, other areas were much less involved. The mesentery was indurated and the mesenteric veins were thrombosed.

GAS IN THE PORTAL VEINS

The final sign of intestinal necrosis is gas in the portal veins as reported earlier.^{1,8,9,11} This sign is seen clearly in Figure 8, a case of mesenteric thrombosis.

In some cases the fine linear shadows of lesser density within the liver may be difficult to observe, especially if the roentgenogram is somewhat underexposed. To overcome this handicap, Gildenhorn *et al.*³ placed their patient in the lateral decubitus position (Fig. 9) and the gas within the portal veins in the liver became far more visible. It should be noted that both intraluminal gas and gas in the portal circulation may be present in the same individual or they may be independent of each other, although both originate from the same source. Gas has been observed in the mesenteric vessels at autopsy, but in the simple roentgenogram of the abdomen it is best demonstrated in the portal vein and its intrahepatic branches.

Gas in the portal veins must be distinguished from gas in the common and intrahepatic biliary ducts resulting from an internal biliary fistula or ascending gas form-

ing cholangitis. Susman and Senturia⁸ have pointed out that the flow of blood in the portal vein is toward the periphery so that the intrahepatic branches of the portal vein may be more visible when filled with gas than the major vein itself. Figure 9, a lateral decubitus study with the right side up, demonstrates this well. The contrary is true in the case of internal biliary fistula, the flow being toward the extrahepatic portion of the common duct and the gallbladder. In many of these cases, the gallbladder will also be seen to contain gas. In others, the large size of the common duct, much broader than the portal vein, and the lack of visualization of the peripheral intrahepatic ducts should make the distinction possible.

There has been considerable discussion as to the source of this gas in the portal circulation. It seems reasonable that the des-

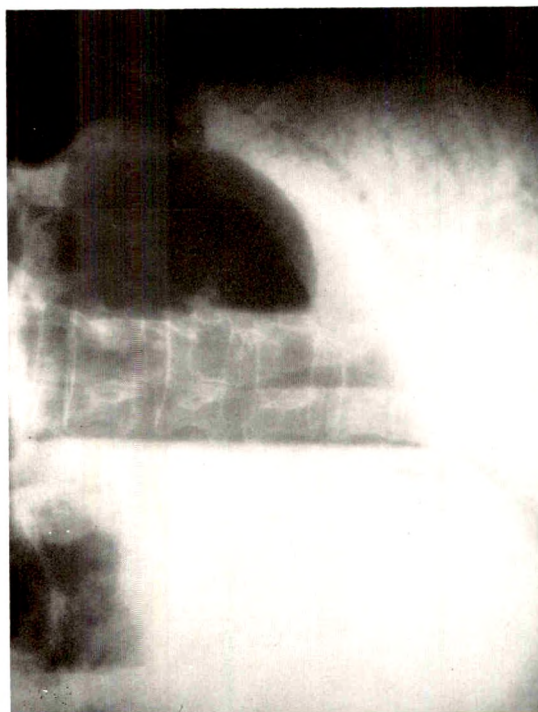


FIG. 9. Case of mesenteric thrombosis. The patient was put in the lateral decubitus position with the right side up so that the gas in the portal circulation was displaced to the periphery of the liver. It is demonstrated here filling the multiple branches of the portal vein.

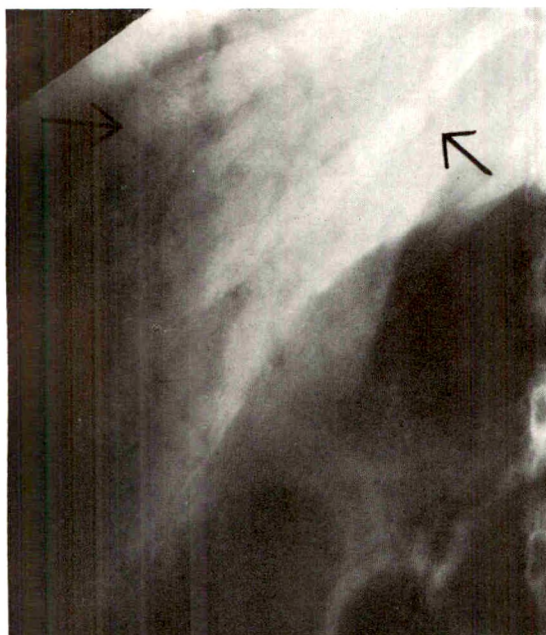


FIG. 8. Case of mesenteric thrombosis with typical signs of gas in the portal circulation. Note the linear shadows of gas density which are narrow and extend well out to the periphery of the liver, producing multiple roughly parallel lines. There is very little gas seen near the medial border of the liver in contrast to the finding in internal biliary fistula in which the major gas shadows are near the medial border in the region of the common duct.

truction of the mucosa would permit direct entrance of gas from the intestinal lumen into the portal circulation. Wiot and Felson,⁹ however, believe that it is due to gas-forming organisms but in our own experience we have not been able to isolate such organisms. In the cases of diabetes which they reported, it is very possible that gas forming bacteria were responsible.

MATERIAL

We have reviewed in cursory fashion some of the acute abdominal cases in 3 hospitals. Many roentgenograms of the abdomen made during the acute episode were examined and we were surprised to find 14 cases which exhibited gas, either in the intramural portion of the bowel, or in the portal veins of the liver, or both. Needless to say, many of these observations were retrospective, since the correct diagnosis was not

TABLE I
CASES WITH INTRAMURAL BOWEL OR PORTAL
VEIN GAS OR BOTH

Case	Intramural Bowel Gas	Portal Vein Gas	Etiology
1	X	X	Mesenteric Thrombosis
2	X		Mesenteric Thrombosis
3	X		Direct Trauma
4	X	X	Necrotizing Gastroenterocolitis
5	X		Mesenteric Thrombosis
6	X		Mesenteric Thrombosis
7	X	X	Mesenteric Thrombosis
8	X		Mesenteric Thrombosis
9		X	Mesenteric Thrombosis
10	X	X	Strangulation Obstruction
11	X		Mesenteric Thrombosis
12	X	X	Perforated Ulcer with Necrosis
13	X		Mesenteric Thrombosis
14	X		Mesenteric Thrombosis

made at the time of the acute episode. The cases found are listed in detail in Table I.

There were 13 cases with gas in the intramural portion of the bowel and 6 with portal vein gas. In only 1 case was there gas in the portal veins without evidence of gas in the wall of the bowel. Even in this case, the evidence is doubtful since the distention of the intestinal tract was so extreme that it was impossible to be certain of the presence of gas outside the lumen of the bowel. However, the gas in the portal veins was clearly seen. As might be expected, there were 8 cases in which gas could be found in the wall of the bowel, but none was observed in the veins. The varying etiologies are listed. The preponderance of mesenteric thrombosis as the cause of the intestinal necrosis is in accordance with the common findings. In 1 case of necrotizing gastroenterocolitis from a severe infection with virulent organisms, gas was found both in the intestinal wall and in the portal veins of the liver. It is notable that Gildenhorn *et al.*³ found no cases with intramural or portal vein gas in the series of necrotizing enteritis cases associated with lymphoma which they reported. Unfortu-

nately, the roentgenograms were made some time before the final stages of the disease. Furthermore, because of the condition of the patient, many of the abdominal roentgenograms were unsatisfactory.

In 1 case, gas in the portal circulation was discovered along with evidences of retroperitoneal gas, as a result of perforation of a gastric ulcer extending into the lesser omental sac. Necrosis of the mesentery with open veins into which apparently the gas from the perforation extended explained this finding. This is the only case we have seen, however, in which gas in the portal circulation has occurred without accompanying clear-cut evidence of intestinal necrosis.

SUMMARY

The roentgen signs of impending or actual intestinal necrosis have been described. The roentgen evidence of intestinal intramural gas reported by Schorr⁷ has been encountered in 9 cases of mesenteric thrombosis, 1 case of perforated gastric ulcer, 1 case of acute necrotizing gastroenterocolitis, 1 case of direct trauma and 1 case of strangulation obstruction. We have also observed gas in the portal circulation in 3 cases of mesenteric thrombosis, 1 case of strangulation obstruction, 1 case of perforated gastric ulcer and 1 case of necrotizing gastroenterocolitis.

The observation of edema of the mucosa of the bowel, of marked distention of a solitary loop with excessive fluid, and of great thickening of the bowel wall with narrowing of the lumen may well signify impending necrosis. The roentgen evidences of gas in the bowel wall and/or gas in the portal veins are highly significant signs of intestinal necrosis.

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We are particularly indebted to Dr. Stanley Lance of the Huntington Memorial Hospital, Pasadena, California, for the privilege of re-

porting Case I (Fig. 6 and 7). We are indebted to Dr. Hyman Gildenhorn of the City of Hope for Figure 9. Figure 1 is from the Cedars of Lebanon Hospital, Los Angeles, California, Figure 8 from St. Mary's Hospital, Long Beach, California.

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THE FLUID-FILLED COLON IN ACUTE LARGE BOWEL OBSTRUCTION*

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THE usual picture of large bowel obstruction on the plain roentgenogram of the abdomen is that of gaseous distention of the colon extending in a retrograde fashion from the point of obstruction to the cecum, often with associated gaseous distention and fluid levels in the small bowel.¹ It is usually possible to determine not only the presence of large bowel obstruction, but also the level at which the obstruction is occurring. There are, however, certain sources of error. Occasionally, the plain roentgenogram may simulate small bowel obstruction with gaseous distention and fluid levels confined to the small bowel, and no distention of the colon. Ulin and colleagues,⁴ in a study of 227 patients with large bowel obstruction, encountered 8 with a "history and physical features leading to a clinical diagnosis (including radiography) of apparently pure small bowel obstruction on admission." Byrne¹ encountered 3 such cases in 143 of large bowel obstruction, and Kent and Raszkowski³ reported 6 such instances. In these cases, the colon roentgenographically appeared to be empty; no mention is made of any soft tissue shadow of fluid-filled colon. Kent and Raszkowski advanced various theories to account for this emptiness, including associated small bowel obstruction and reversed peristalsis emptying the colon. Williams⁶ has recently drawn attention to a second source of error, namely, that fluid alone may accumulate in the bowel in simple obstruction in a similar fashion to the accumulation of fluid in a strangulating obstruction. There is little or no gas in these fluid-distended loops and the supine roentgenogram may be considered normal. The erect roentgenogram may be con-

sidered to show minimal abnormality, with only a solitary gas-distended loop, unless the soft tissue outline of the fluid-filled bowel loops is appreciated. He described this change as occurring in small bowel obstruction, but he mentioned its occurrence in 3 cases of colonic obstruction.

We have in the last 18 months encountered 6 cases illustrating another source of possible error, in that there may be fluid accumulation in the colon proximal to the point of obstruction, extending in a retrograde fashion possibly as far as the cecum, with gaseous distention of the bowel proximal to the fluid-filled loops. On the plain roentgenograms of the abdomen, therefore, the marked gaseous distention suggests intestinal obstruction, but unless the soft tissue outline of the fluid-filled colon is appreciated, the point of obstruction is erroneously placed at the level of the termination of the gaseous distention. This phenomenon has received little attention hitherto in the literature. Frimann-Dahl² makes passing mention of the fact that in obstruction of the transverse colon, the cecum and ascending colon may be distended by fluid alone in this fashion, producing a soft tissue shadow without gaseous distention. Williams⁶ also draws attention to the phenomenon of accumulation of fluid proximal to an obstruction of the right half of the colon with gaseous distention confined to the small bowel and the roentgenologic picture therefore suggesting small bowel obstruction. The 6 cases that we are reporting are of interest not only in that they illustrate this phenomenon of fluid-filling of the colon with air distention of bowel proximal to the fluid-filled loops, but also that in all these cases, the obstruction

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occurred in the distal colon. In 2 cases the gaseous distention was confined to the small bowel, suggesting small bowel obstruction. In the other 4, the gaseous distention involved the colon, but the level of termination of the distention suggested obstruction at the wrong level.

REPORT OF CASES

CASE I. This 46 year old female was admitted with a history of chronic constipation for 12 years, and progressive colicky abdominal pain, nausea and absolute constipation for 5 days.

A supine roentgenogram of the abdomen (Fig. 1A) showed considerable gaseous distention of the cecum and ascending colon and of a remarkably redundant transverse colon. The gaseous distention ended abruptly at the splenic flexure. There was a little scattered air in the small bowel. The distribution of gas suggested an obstruction at the splenic flexure, but the soft tissue shadow of fluid-filled descending colon could be seen extending down into the pelvis. An erect roentgenogram showed a similar distribution of gas with fluid levels in the ascending and transverse colon, but no fluid level in the splenic flexure or descending colon. The soft tissue outline of the fluid-filled descending colon could again be clearly made out.

An emergency barium enema study was performed, and this revealed a malignant obstruction of the distal sigmoid colon. A certain amount of barium passed beyond the obstruction, and this showed even more clearly the extent of the fluid-filled loop visible on the plain roentgenograms (Fig. 1B).

At subsequent operation, an annular adenocarcinoma was found constricting the sigmoid colon.

CASE II. This 62 year old woman was admitted with a history of crampy abdominal pain and absolute constipation for 3 days, and with vomiting for 24 hours.

The supine roentgenogram of the abdomen on admission (Fig. 2A) showed a moderate gaseous distention of the distal small bowel and gross distention of the cecum, ascending colon and transverse colon ending sharply at the splenic flexure and suggesting obstruction at this level. The soft tissue outline, however, of the fluid-filled descending colon could be seen running from the splenic flexure down to the rim of the true pelvis. An erect roentgenogram

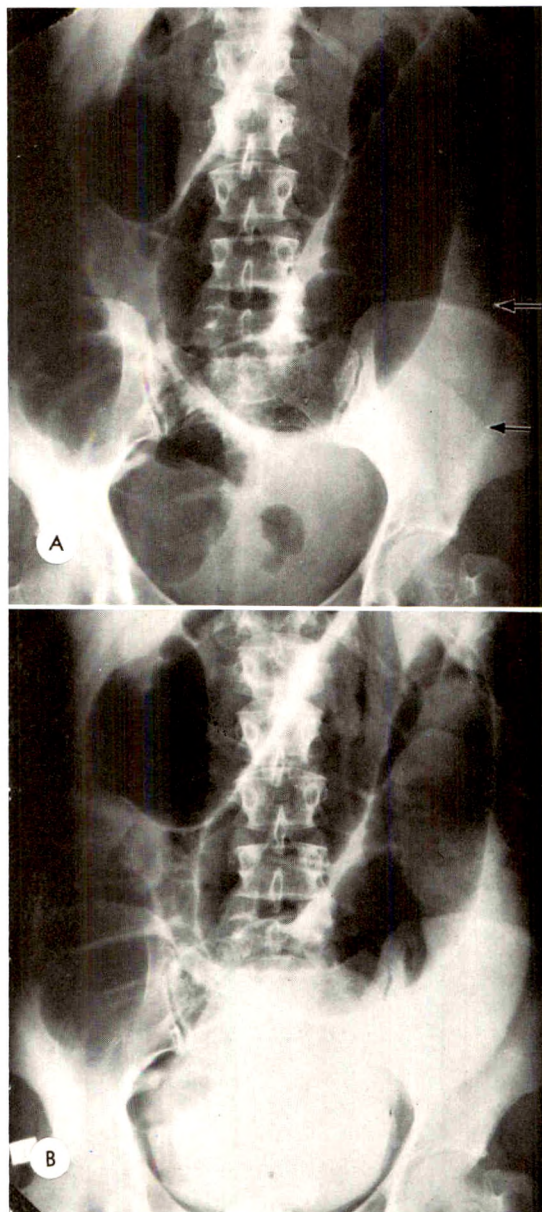


FIG. 1. Case I. Carcinoma of sigmoid colon. (A) Supine roentgenogram showing gaseous distention suggesting obstruction at the splenic flexure. Soft tissue outline of fluid-filled descending colon ($\leftarrow$). (B) Supine roentgenogram after barium enema. Fluid-filled descending colon opacified by barium.

(Fig. 2B) showed a similar distribution of intestinal gas with fluid levels, except that some gas had now been displaced into the upper descending colon with a fluid level below it. The soft tissue outline of the fluid-filled descending colon could again be seen.

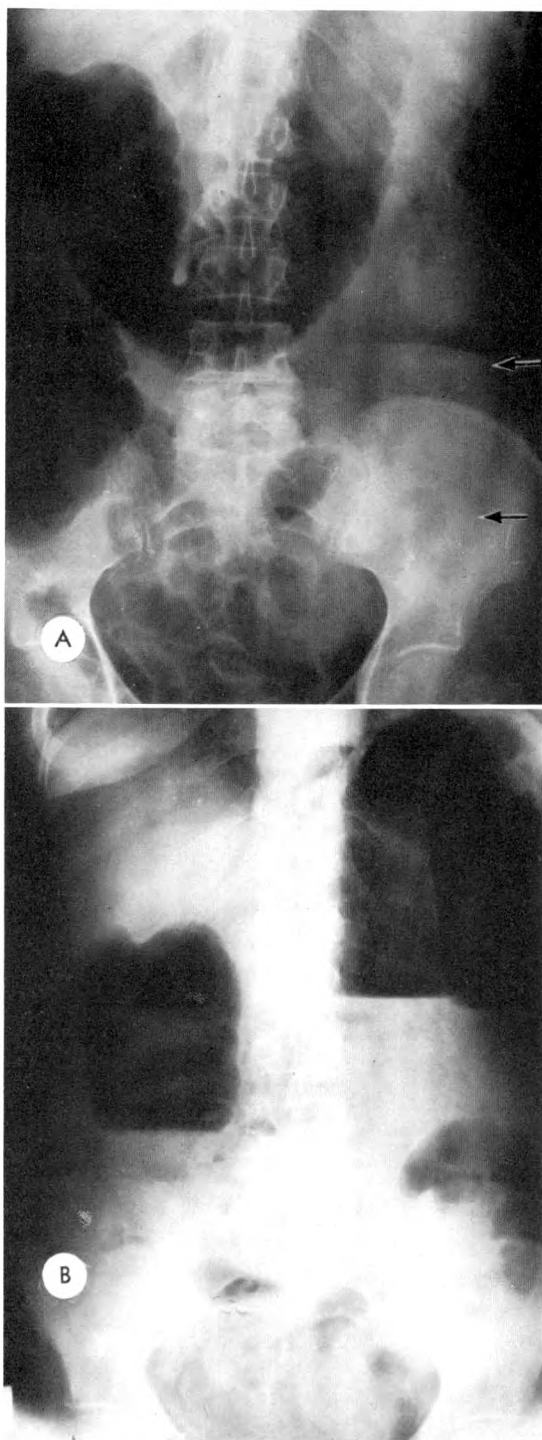


FIG. 2. Case II. Carcinoma at junction of sigmoid and descending colon. (A) Supine roentgenogram showing gaseous distention suggesting obstruction at splenic flexure. Soft tissue outline of fluid-filled descending colon ($\leftarrow$). (B) Erect roentgenogram. Fluid level in upper descending colon. Soft tissue shadow of descending colon is still visible.

The gaseous distention on the supine roentgenogram in this case would suggest obstruction at the splenic flexure, but the erect roentgenogram would indicate obstruction at a lower level. The shadow of the fluid-filled colon, however, would give strong indication of obstruction in the pelvic colon, and a barium enema study showed an obstructing carcinoma in the pelvis, at the junction of sigmoid colon with descending colon. This finding was confirmed at surgery.

CASE III. This 41 year old man was admitted with a 2 day history of crampy abdominal pain and constipation, and with nausea for 24 hours.

A supine roentgenogram of the abdomen (Fig. 3) showed some gas in the right half of the transverse colon, but nowhere else in the colon, and a marked gaseous distention of the jejunum with a little air in the ileum. The distribution of gas suggested a partial small bowel obstruction, but the soft tissue outline of fluid-distended ascending and descending colon could be seen in both flanks, on the left side extending down into the true pelvis. An erect roentgenogram showed considerable gaseous distention with fluid levels in the jejunum

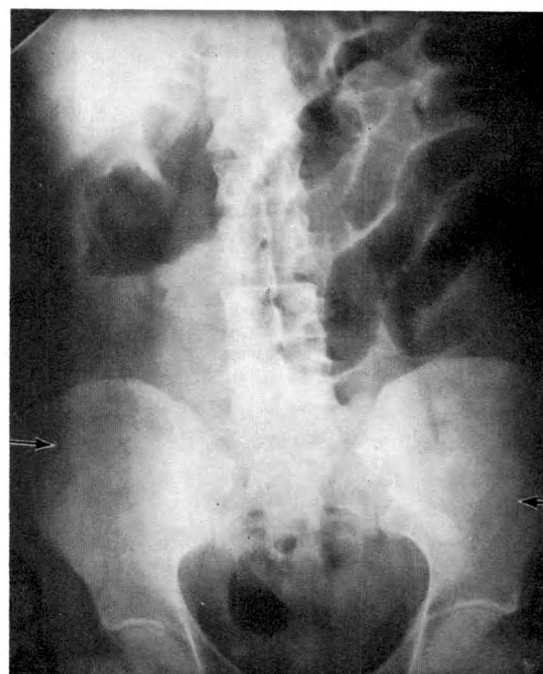


FIG. 3. Case III. Carcinoma at junction of descending and sigmoid colon. Supine roentgenogram suggesting small bowel obstruction, although there is air in the right transverse colon. Bilateral soft tissue masses of fluid-filled ascending and descending colon ($\rightarrow\leftarrow$).

and to a lesser extent in the ileum with an air-fluid level in the hepatic flexure, but no air-fluid elsewhere in the colon. The soft tissue outline of the fluid-filled descending colon could again be well made out.

A barium enema study showed a malignant obstruction at the junction of the descending and the sigmoid colon, and at operation, an annular carcinoma was found.

CASE IV. This 40 year old female was admitted with a vague history of some difficulty in defecation for the past 2 months, and nausea, vomiting and absolute constipation for 48 hours.

A supine roentgenogram of the abdomen (Fig. 4) showed a considerable gaseous distention of the small bowel, particularly of the jejunum, but no distention of the colon. There was probably some air in the hepatic flexure. The appearance suggested a small bowel obstruction, but the soft tissue outline of both fluid-distended ascending and descending colon could be made out. An erect roentgenogram showed multiple air-fluid levels in the small bowel; there was some air in the transverse colon, which was overlain by air-filled small bowel loops.

This patient was submitted to surgery without a preliminary barium enema study, and an adenocarcinoma was found obstructing the bowel at the junction of descending and sigmoid colon.

CASE V. This 39 year old female was admitted with a history of attacks of colicky abdominal pain for 1 year, increasing constipation for 3 weeks, and absolute constipation, abdominal pain, distention and vomiting for 24 hours.

A supine roentgenogram of the abdomen (Fig. 5A) showed a moderate gaseous distention of the small bowel with considerable distention of the cecum and ascending colon and a less marked distention of the proximal two-thirds of the transverse colon. The transverse colon was redundant and took a long loop towards the pelvis. There was no distention of the distal third of the transverse colon, and no air could be seen in the descending colon. The appearance suggested an obstruction of the transverse colon, but the soft tissue shadow of the fluid-filled colon could be made out extending into the pelvic bowl.

An erect roentgenogram (Fig. 5B) showed similar distention with fluid levels in the small bowel and proximal colon, with, in addition, an air-fluid level in the splenic flexure. An

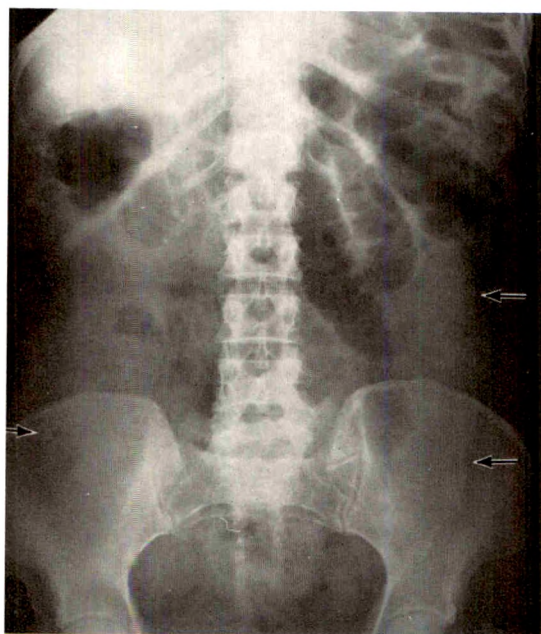


FIG. 4. Case IV. Carcinoma at junction of descending and sigmoid colon. Supine roentgenogram suggests a small bowel obstruction. Some air in hepatic flexure. Bilateral soft tissue masses of fluid-filled ascending and descending colon ($\rightarrow \leftarrow$).

emergency barium enema study showed a malignant obstruction at the apex of the sigmoid loop and at operation an annular adenocarcinoma was found at this site.

CASE VI. This 51 year old female was admitted with a 2 day history of colicky abdominal pain and absolute constipation. A supine roentgenogram (Fig. 6A) showed scattered air in the small bowel, with marked gaseous distention of the cecum and colon terminating abruptly at the splenic flexure. The appearance was suggestive of obstruction at the splenic flexure, but the soft tissue outline of descending colon extending into the pelvis could be seen.

An erect roentgenogram (Fig. 6B) showed a similar distribution of gas, with an air-fluid level in the proximal limb of the splenic flexure. The soft tissue outline of the descending colon could, however, again be made out. A barium enema study showed a malignant obstruction of the proximal sigmoid colon and at operation an adenocarcinoma was found.

CONCLUSION

In many centers, the routine initial examination in cases of suspected intestinal

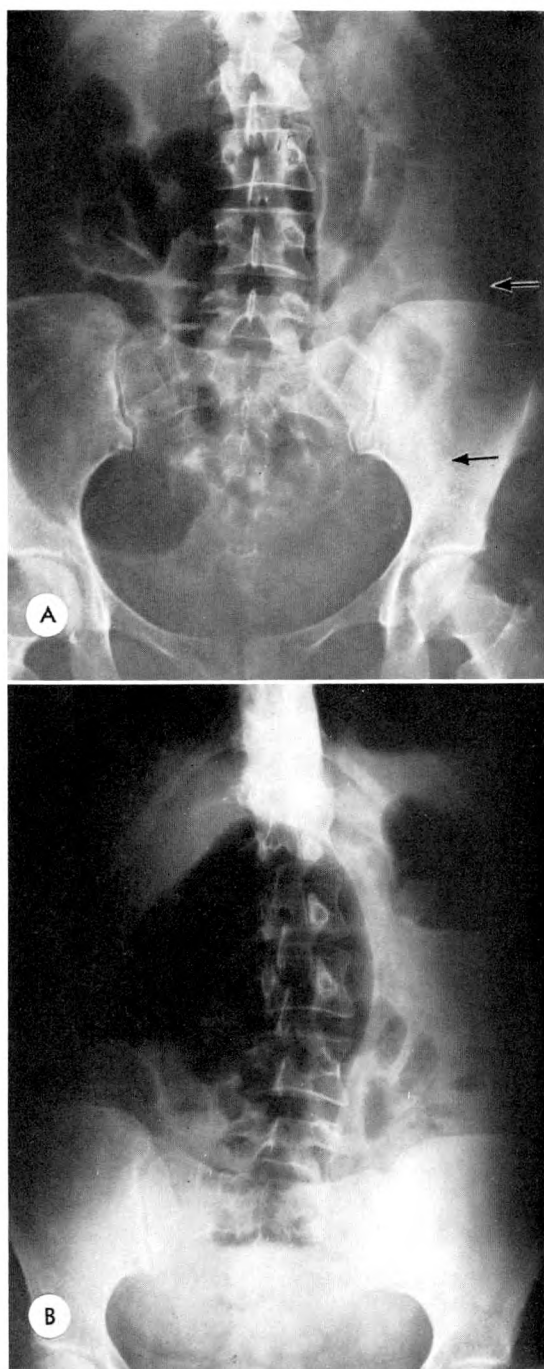


FIG. 5. Case v. Carcinoma at apex of sigmoid loop. (A) Supine roentgenogram suggesting obstruction of distal transverse colon. Soft tissue mass of fluid-filled descending colon ($\leftarrow$). (B) Erect roentgenogram. Fluid level in splenic flexure. Fluid-filled descending colon again visible.

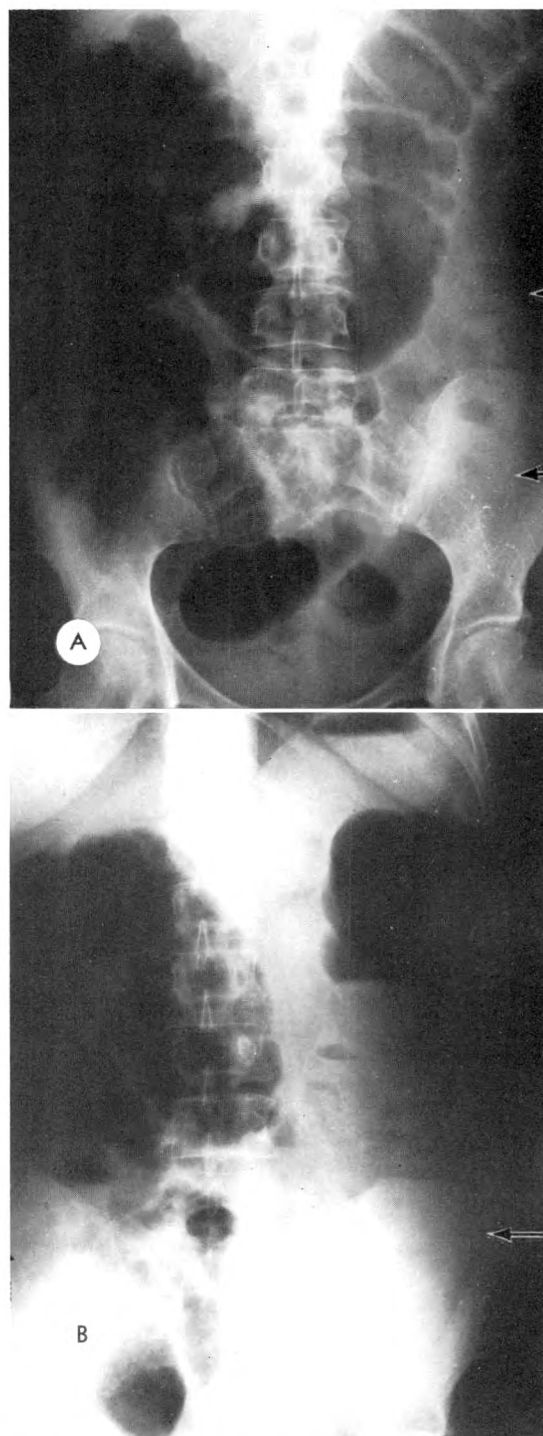


FIG. 6. Case vi. Carcinoma of proximal sigmoid colon. (A) Supine roentgenogram suggesting obstruction at splenic flexure. Fluid-distended descending colon ($\leftarrow$). (B) Erect roentgenogram. Fluid-filled descending colon again visible ($\leftarrow$).

obstruction consists of a supine and an erect roentgenogram of the abdomen. The 6 cases presented here would suggest that in those instances where the termination of the gaseous distention is misleading, the supine roentgenogram alone may well give strong indication of the actual level of obstruction in the distal colon, if the sign of the fluid-filled colon is appreciated and looked for. If the examination is supplemented by an erect roentgenogram, little further information is provided as to the level of obstruction in those cases in which the gaseous distention is confined to the small bowel; in those with air in the proximal colon, the appearance of a fluid level in the upper descending colon together with the persistence of the soft tissue mass of the fluid-filled descending colon will add to the suspicion of the distal colonic obstruction. It seems unlikely that left lateral decubitus roentgenograms with a horizontal ray will contribute further information; they were performed in only 2 cases of the present series and were noncontributory. In those cases where gaseous distention is confined to the small bowel, it is unlikely that a right lateral decubitus roentgenogram would help, but in those with gaseous distention extending to the splenic flexure, it is likely that a right lateral decubitus roentgenogram would show a horizontal fluid level in the descending colon. The chief value of this sign would, therefore, seem to lie in those cases mimicking small bowel obstruction, and in those cases of misleading gaseous distention of the colon where no right lateral decubitus roentgenogram is available.

SUMMARY

Six cases of obstruction of the distal colon are presented in which plain roentgenograms showed gaseous distention of the bowel terminating well proximal to the point of obstruction of the colon, in 2 cases the gaseous distention being almost entirely confined to the small bowel and mimicking small bowel obstruction. This is shown to be due to distention of the colon immediately proximal to the obstruction by fluid rather than by air. Unless the appearance of the soft tissue mass of the fluid-filled colon is appreciated, the level of the obstruction is liable to be misdiagnosed roentgenologically.

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ABDOMINAL AORTIC AND ILIAC GRAFT FISTULAE*

UNUSUAL ROENTGENOGRAPHIC FINDINGS

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GRAFT replacement for abdominal aneurysms is not an uncommon procedure. DeBakey *et al.*² recently reported a follow-up study of 1,719 such cases.

Varying incidences of graft failures have been reported by different authors. Vasko and his co-workers¹⁹ had 6 failures out of 123 patients, due to rupture of the graft or recurrent aneurysm. Crawford *et al.*,¹ in a review of the experience at Baylor, reported 6 aorto-duodenal fistulae in 638 cases of abdominal aortic grafts. Altogether, there have been 56 cases of abdominal aortic graft-intestinal fistulae reported in the world literature.^{4,8} Forty-five were aortoduodenal fistulae and 11 occurred in the jejunum or ileum. According to Harrison,¹⁰ infection at the suture line is the cause of most graft failures. Furthermore, the predilection of graft rupture as well as aneurysm rupture for the third portion of the duodenum is probably related to the immobility of this segment, thus facilitating erosion by constant pulsatile pressure at a stationary part.

Survival after graft rupture into the intestine is unusual. Erskine *et al.*⁵ in their review of the literature up to 1960, found only 5 survivors of 24 reported cases. In 1958, MacKenzie and his colleagues¹³ reported the first case of survival after resection of an infected homograft with rupture into the gastrointestinal tract. Numerous reports have appeared in the literature concerning repair of graft-intestinal fistulae.¹⁸

While the clinical symptomatology of rupture at the anastomotic site is well known (triad of shock, pain, and palpable mass),⁹ there has been very little in the literature concerning the roentgenographic findings of graft-intestinal fistula forma-

tion. Therefore, 3 cases of graft-intestinal fistulae are reported with emphasis on 3 different roentgenographic findings.

REPORT OF CASES

CASE 1. Boston City Hospital #60817. S.O., a 42 year old male with a history of graft replacement (teflon) of the mid-abdominal aorta, 2 years prior to admission, entered the hospital with massive gastrointestinal bleeding.

Roentgenographic Findings. An upper gastrointestinal series demonstrated a compression defect of the third portion of the duodenum (Fig. 1). The right anterior oblique study showed a streak of barium conforming to the wall of the graft (Fig. 2 and 3). The lace-like pattern of the tubular graft was apparent.

Course. At surgery, a pin-point fistula was found between the duodenum and the aortic graft at the site of graft-vessel anastomosis. No infection was noted. Since complete replacement of the graft was not felt to be indicated, a mobilization patch procedure was performed. The patient recovered, but recurrence of a fistula 3 months later necessitated replacement of the entire graft. At this time, the patient is asymptomatic.

Comment. Considering the history in this case, as well as the roentgenologic findings, it is difficult to consider an alternative diagnosis. A fistula or long sinus tract from a perforated duodenum would be a remote possibility. Of interest was the unique appearance of the barium around the graft. This collection disappeared in 24 hours. Its initial persistence and lack of free flow were probably due to a sinus tract from the duodenum to the fibrous sheath of the graft, rather than to a true fistula. There was no evidence of barium in the graft when it was resected some 3 months later. In this case, the graft dilated at the graft-

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vessel suture site, but there was no evidence of infection.

CASE II. Boston City Hospital #60838. P.K., a 68 year old male, entered the hospital with acute massive gastrointestinal bleeding. There was a history of a peptic ulcer and a gastrojejunostomy approximately 10 years ago. Eighteen months previously, a graft was inserted for a large mid-abdominal aortic aneurysm.

Roentgenographic Findings. An upper gastrointestinal series showed incomplete obstruction of the duodenum at the junction of the third and fourth portion with dilatation proximally (Fig. 4).

Course. The patient's shock could not be controlled and he expired before surgery could be attempted. At postmortem examination,



FIG. 1. Case I. Upper gastrointestinal study shows compression at the third portion of the duodenum (arrow). The markedly dilated small bowel may be secondary to vascular insufficiency.



FIG. 2. Case I. Roentgenogram in the right anterior oblique projection clearly demonstrates the tubular graft outlined with barium (arrows).

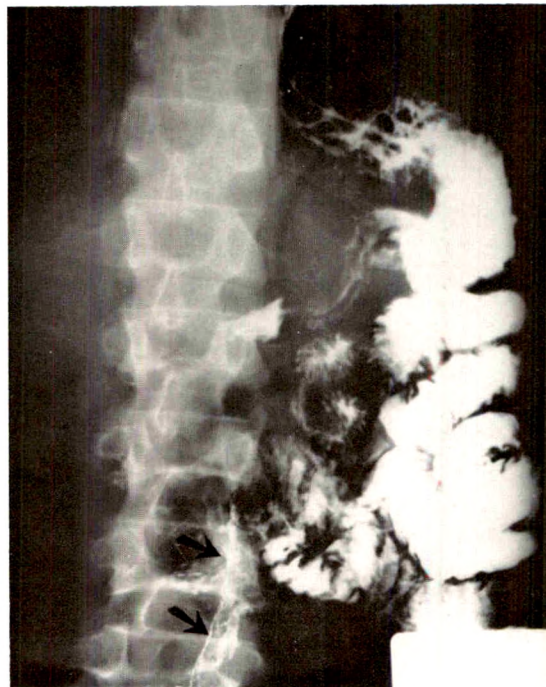


FIG. 3. Case I. Anteroposterior roentgenogram shows the outlined graft overlying the lower lumbar spine (arrows).

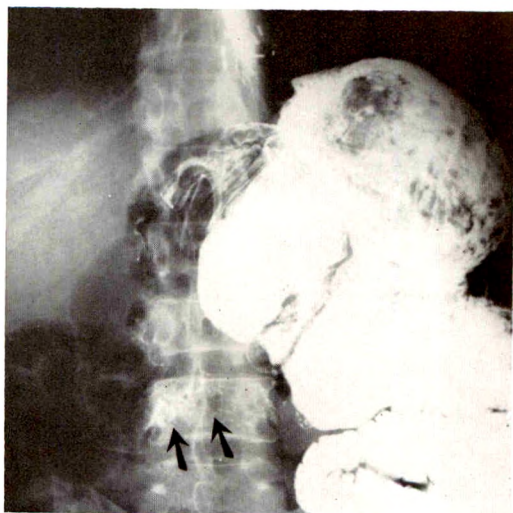


FIG. 4. Case II. Upper gastrointestinal study shows in the anteroposterior projection a sharp cut-off at the third portion of the duodenum (arrows). The radiolucent mottling in the stomach is probably due to blood clot. Note the gastrojejunostomy functioning as an overflow valve in this particular case.

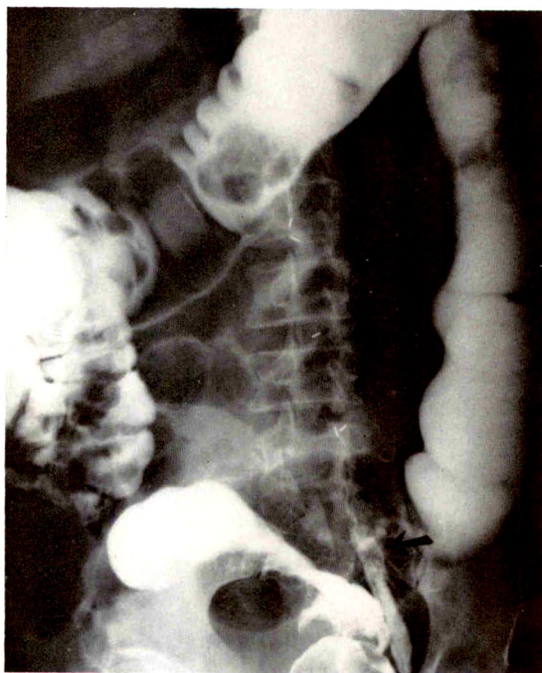


FIG. 5. Case III. Barium enema study shows a soft tissue mass in the pelvis with a pressure deformity of the sigmoid colon. The barium filled iliac graft is seen communicating with the sigmoid colon (arrow).

the graft was noted to have ruptured into the duodenum at the site of vascular-graft anastomosis. Microscopic sections showed foci of inflammation, but cultures for bacteria were negative.

Comment. A similar cut-off at the third and fourth portion of the duodenum can be seen in vascular mesenteric root compression, pancreatitis and peptic ulcer disease. In this particular case, we were unable to determine the exact etiology of the dilated proximal duodenal loop. The gastrojejunostomy, it would seem, functioned as an overflow valve, thereby preventing marked obstruction.

CASE III. Boston City Hospital #2018208. T.L., a 50 year old white male, had an ilio-femoral bypass graft operation in April, 1962, for left iliac occlusive disease. He did well until May, 1964, when he developed an aneurysm in the graft at the femoral junction. This was repaired with a dacron patch. However, sepsis developed at the patch site with ultimate formation of an abscess in the left groin. At surgery, the noted breakdown of the graft neces-

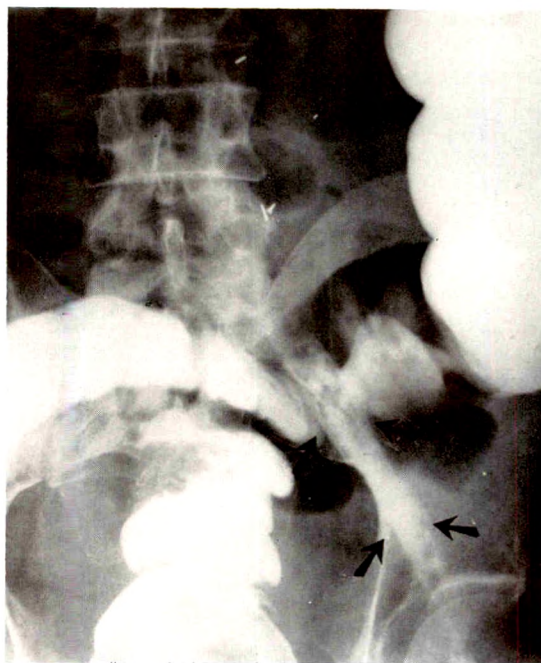


FIG. 6. Case III. Close-up view of Figure 5 shows the iliac graft fistula to better advantage (arrows).

sited its ligation at the proximal portion with an above the knee amputation. A drain was inserted into the abscess site. Some 3 days later, after a stormy febrile course, fecal material was noted at the abscess site with free drainage.

Roentgenographic Findings. A barium enema examination showed opaque material in the iliac bypass graft on the left (Fig. 5 and 6). There was an associated pelvic mass with stretching and displacement of the sigmoid colon.

Course. Colostomy was done, but no further pelvic surgery was performed. It appears that the infection of the graft was the etiology of the fistula here.

Comment. The roentgenographic picture in this case was considered to be unique. Certainly, the absence of flow into the inferior vena cava and resultant barium pulmonary emboli rule out sigmoid vein fistula. The caliber of the tubular collection and its flow distally militate sufficiently against a colonic-lymphatic fistula. The tubular collection demonstrated on roentgenograms does not resemble a nonspecific groin fistula.

DISCUSSION

Although rupture of grafts into the intestine is a well known occurrence, the roentgenographic findings are less well known. The 3 cases described represent different facets of the same process.

In Case I, the streak of barium was, we presume, in the fibrous covering of the aorta. This would seem to be a reliable sign of graft-duodenal fistula formation. However, in this particular case, the lack of free lumen (graft) communication is diagnostic of a sinus tract rather than a true fistula.

In Case II, the pressure deformity on the third portion of the duodenum is a common sign of abdominal aneurysm.¹⁶ However, complete or even partial obstruction is unusual, only 8 cases having been reported.^{3,6,11,14,15,17,20} The roentgenographic picture of pressure deformity of the third portion of the duodenum in our case mimics the partially obstructive duodenal adherence of an aneurysm. The clinical history and roentgenographic findings seem

to point toward graft adherence with or without rupture.

In Case III, we feel that the circumstances surrounding the occurrence of the graft-colonic fistula are unusual. We are aware of only 2 aneurysm ruptures into the colon,^{7,12} and were unable to find a report of a graft-colonic fistula in the literature. We believe that our case is unique. It is difficult to suggest any differential diagnosis in this case in view of the described roentgenographic picture. Ureter, vein, lymphatic, or nonspecific fistulae have already been commented upon.

With the number of vascular graft replacements being performed today, we feel that awareness of the described findings is of value in the roentgenographic diagnosis.

SUMMARY

Three cases of graft-intestinal fistulae are reported. The roentgen findings are: opaque medium in the fibrous sheath, pressure erosion with fistula formation, and partial obstruction of the third portion of the duodenum, as well as a sigmoid fistula.

Familiarity with these findings may be of value in the roentgenographic diagnosis of vascular graft-intestinal fistulae. Prompt and correct detection of such fistulae should reduce the mortality rate in these cases.

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PROSTATIC CARCINOMA INVOLVING THE RECTUM AND SIGMOID COLON*

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CARCINOMA of the prostate can extend posteriorly and involve the rectum to such an extent as to simulate primary rectal and sigmoid disease on physical and roentgen examinations. The differentiation of this rectal involvement by prostatic carcinoma from that of primary colon carcinoma is essential, since the therapy is quite different for the two entities: the rectal carcinoma may require an abdominoperineal resection; the invasive prostatic carcinoma is treated by orchiectomy and/or hormone therapy. Extensive prostatic carcinoma is not considered a "surgical lesion" since it is generally beyond the realm of total excision by radical surgery and, therefore, is treated by chemotherapy. An extensive surgical procedure performed in the case of an erroneous diagnosis of "rectal cancer" is unfortunate, and offers no hope of salvage from an extensive malignancy that is already metastatic.

STAGING

Clinically, the involvement of the colon is divided into 3 types:⁸

1. *An anterior rectal mass* (Fig. 1A). There is an anterior tumor mass starting in the site of the prostate gland and frequently extending proximally. The over-all size of the mass may not be appreciated by palpation. The passage of the sigmoidoscope is commonly obstructed by the mass on the anterior wall of the colon. The rectal mucosa is not involved by the malignant invasion, but may be edematous. The patient may have symptoms of bloody or mucoid diarrhea, and a change in bowel habits associated with the narrowing of the rectal lumen does occur.

2. *An annular rectal stricture* (Fig. 1B).

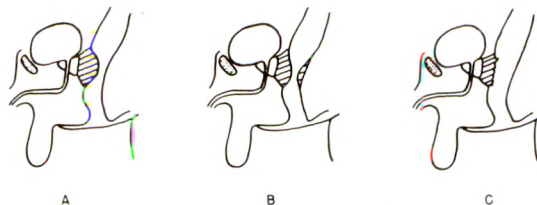


FIG. 1. (A) The anterior mass is a direct posterior extension of the prostatic carcinoma. The mucosa is intact. (B) The stricture is annular and may extend proximally to involve the sigmoid. As in A, the mucosa is intact. (C) The anterior mass has invaded the bowel mucosa. Ulceration is frequent. (Adapted from Davis⁸ with permission of the Livingstone Company.)

The rectum is stenosed circumferentially and, as in Type 1, there is no evidence of mucosal invasion. Here also, endoscopy may show the mucosa to be edematous. The area of involvement is usually long and the margins of the involved colon are tapered. Constipation is a common complaint and obstipation may occur.

3. *An anterior rectal mass and ulcer* (Fig. 1C). In addition to a tumor mass, the mucosa in this instance is invaded by the neoplastic extension and ulceration is present. This lesion is the most similar in appearance to primary rectal carcinoma.

CLINICAL PATHOLOGIC FEATURES

Twenty per cent of males with prostatism have carcinoma of the prostate, independent of age.⁸ It has been found at autopsy that 20 to 25 per cent of males over 50 years of age have carcinoma of the prostate,¹¹ irrespective of clinical findings, and postmortem examination shows that approximately 14 per cent of the carcinomas of the prostate have extended posteriorly in varying degrees to involve the rectum.⁹ Car-

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TABLE I
INCIDENCE OF COLON INVOLVEMENT IN
CARCINOMA OF THE PROSTATE

	Total No. of Cases	Colon Involve- ment	Per Cent
Graves and Militzer ⁵	120	5	4.2
Kickham ⁷	132	12	9.1
Young ¹³	800	12	1.5
Arnheim ¹	176	11	6.3
Winter ¹¹	150	13	8.7
Winter ¹²	225	26	11.5
Becker	334	11	3.3

cinoma of the prostate develops in the posterior segment of the gland in 75 to 80 per cent of the cases.² Histologically, the tumor, even when a few millimeters in size, invades the capsule.⁹ This may well explain why such a high percentage of patients with carcinoma of the prostate have incurable disease when first seen.

Three courses of growth after the extension through the prostatic capsule may occur, corresponding to the clinical stages. After the posterior breeching of the capsule, the tumor apparently encounters little resistance from Denonvilliers' fascia (a fascial plane between the prostate and rectum). There is no peritoneum between the rectum and prostate. A localized tumor mass may extend directly posterior to the prostate and, therefore, an anterior rectal mass is found. The rectum is not invaded and is only displaced posteriorly. A second and alternative course is the circumferential stricture. This occurs because the metastatic path to direct mucosal invasion is blocked. A possible explanation is the fact that Denonvilliers' fascia consists of 2 layers. Penetration of the periprostatic layer is easy and occurs early; once in the potential space between the 2 layers, it may be easier for the tumor to spread laterally and encircle the bowel than to penetrate the second, prerectal layer, and invade the colon directly. With extensive involvement, the malignancy will obstruct the colon; complete obstruction may neces-

sitate an emergency colostomy.⁸ The third alternative route is spread through the prerectal Denonvilliers' fascia into the bowel wall. This is quite rare. Since the mucosa becomes involved, an ulcer of the colon occurs frequently.

MATERIAL

Three hundred and thirty-four patients were admitted to Temple University Hospital in a 10 year period from 1954 to 1963 with a histologic diagnosis of prostatic carcinoma made during their hospitalization. Eleven of these patients were found to have colon involvement. This incidence of 3.5 per cent is low in comparison to previously reported studies (Table I). Our patients with prostatic carcinoma were not routinely evaluated for rectal involvement, as they were in the series reported by Winter.¹¹ This may explain the low incidence, since some of the patients may not have had colonic symptoms. Ten of the 11 patients had symptoms referable to the urinary tract or colon. Thus, the colon was examined either with a sigmoidoscope or barium enema studies, or both. The findings, in general, were quite advanced in the colon. A small mass lesion of the colon rarely produced symptoms, since the rectal mucosa is not involved in a great majority of cases. Therefore, only advanced colon involvement causing obstruction symptoms would draw the attention of the clinician to the colon. Only with a mass of significant size did the patient complain of change in bowel habits, such as constipation. None of the patients in this series complained of diarrhea or rectal bleeding secondary to malignant colon involvement.

ANTERIOR RECTAL MASS

Of the 11 patients, 30 per cent presented with an anterior rectal mass. Two of the lesions were small in size and were only discovered as part of the clinical evaluation of the patient.

CASE I. This 63 year old male was first seen at Temple University Hospital in August of 1962

with the chief complaint of pain in the base of the spine. There were no significant symptoms referable to the gastrointestinal tract. The rectal examination demonstrated a hard prostate gland with a probable mass in its left lobe. Laboratory examination revealed an acid phosphatase of 35.5 King-Armstrong units and an alkaline phosphatase of 21.5 King-Armstrong units. Roentgen examinations revealed a normal intravenous urogram, and diffuse metastatic lesions of the spine. A small anterior rectal mass was demonstrated on barium enema examination (Fig. 2). The patient was treated by a transurethral resection, orchiectomy, and with stilbesterol. He was discharged from the hospital and returned 4 months later with severe headaches, nausea and vomiting of recent onset. At this time the rectal examination revealed a prostate gland which was tender and enlarged. The acid phosphatase was 3.1 King-Armstrong units and the alkaline phosphatase 13.9 King-Armstrong units. The patient was treated symptomatically and discharged. No further follow-up is available.

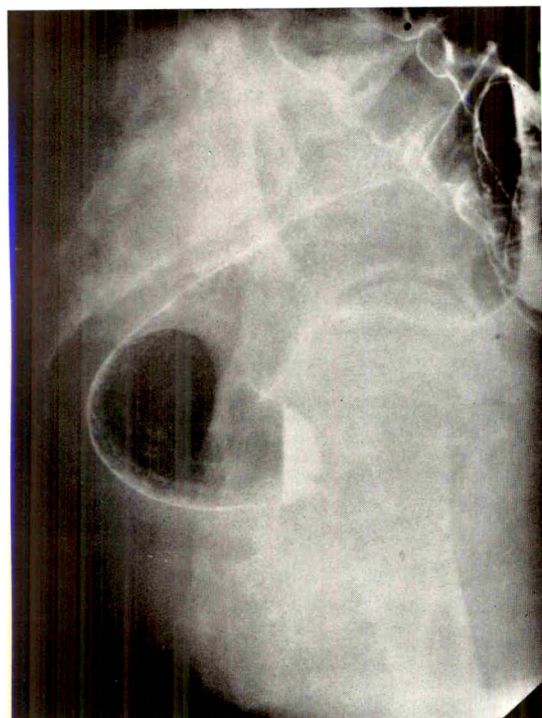


FIG. 2. Case 1. A small anterior extrinsic pressure effect is present. This is best demonstrated with the patient prone and a horizontal roentgen-ray beam used for the exposure.

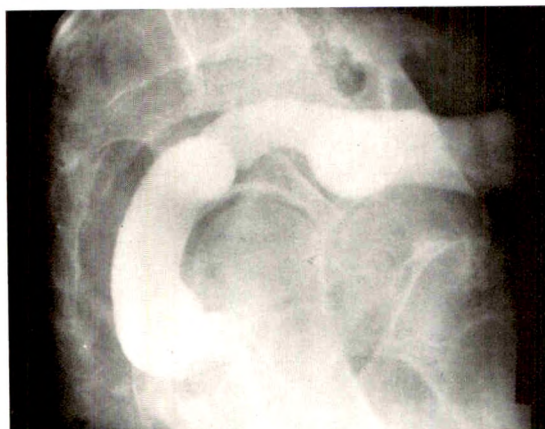


FIG. 3. The small prostatic mass, similar to that in Figure 2, is the result of benign prostatic hypertrophy. The small anterior mass should suggest the possibility of a prostatic carcinoma (since rectal findings are uncommon in benign hyperplasia) but is in no way diagnostic of such.

The pathologic significance of a small anterior rectal mass is not definite. In the usual lateral projection and, especially, with a prone horizontal beam lateral roentgenogram, a small extrinsic pressure effect upon the rectum can be seen in benign prostatic hypertrophy. A patient with this diagnosis (Fig. 3) demonstrated an appearance of the anterior rectal mass similar to that of the patient reported above. Although only a transurethral resection was done in this instance and the entire prostate was not removed, it is felt that an extensive process producing rectal findings would have invaded anteriorly, so that on transurethral resection, sufficient prostatic tissue would be obtained to confirm or exclude the diagnosis of prostatic carcinoma. The conclusion must be drawn in this instance that the finding of a small anterior mass may not be of clinical significance in the diagnosis of prostatic carcinoma and may merely represent benign hypertrophy.

ANNULAR RECTAL STRICTURE

The most common mode of involvement of the colon by prostatic carcinoma is the annular stricture. Seventy per cent of the patients in this series and the majority of reported patients in a review of the litera-

TABLE II
TYPE OF COLON INVOLVEMENT

Authors	Anterior Mass	Annular Stricture	Rectal Ulcer	Other	Total Cases
Baum and McClellan ²	0	5	0		5
Jackman and Anderson ⁶	14	2	11		27
Winter ¹¹	2	6	3	2	13
Winter ¹²	8	13	7	4	26
Davis ³	3	11	2		16
Becker	4	7	0		11

ture had encircling rectal lesions (Table II). This, most likely, is secondary to the ease with which the colon is surrounded. The presenting symptoms in these patients are quite variable. A patient in this group had symptoms of hemorrhoids, and a barium enema examination was done as a routine screening procedure prior to surgery.

CASE II. This 76 year old male was admitted to Temple University Hospital with complaints of pruritus ani and bright red rectal bleeding. No other bowel symptoms were present. In particular, he did not complain of constipation. The physical examination revealed prolapsed internal hemorrhoids. A routine barium enema study showed complete obstruction to the rectograde passage of barium. There was a smooth taper to the colonic narrowing, contrary to the

usual cutoff secondary to rectal carcinoma (Fig. 4, A and B). Skeletal roentgenograms failed to reveal any evidence of metastatic bone disease. The acid phosphatase was 1.3 Bodansky units. A transurethral resection was done and tissue from this was reported as adenocarcinoma of the prostate.

The majority of patients, however, did have symptoms referable to the colon: constipation and/or abdominal pain. These were frequently associated with symptoms of prostatism such as hesitancy, dysuria, and frequency.

CASE III. This 70 year old Negro male was admitted to Temple University Hospital with a history of frequency and urgency of 1½ months' duration. On physical examination a large

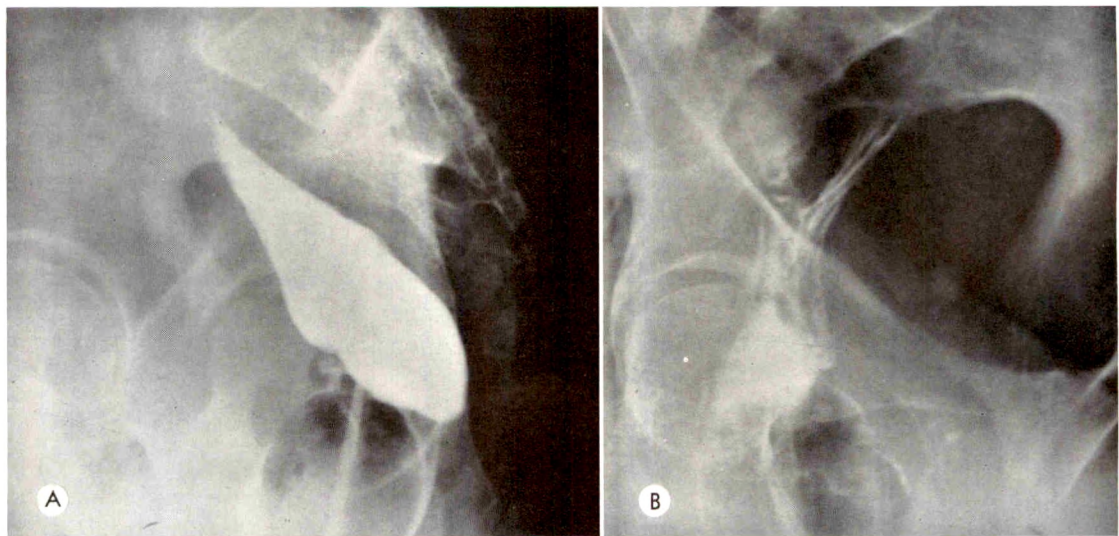


FIG. 4. Case II. (A and B) There is an obstruction to the rectograde passage of barium at the rectosigmoid. The narrowing is tapered in character, unlike that of a primary, encircling carcinoma.

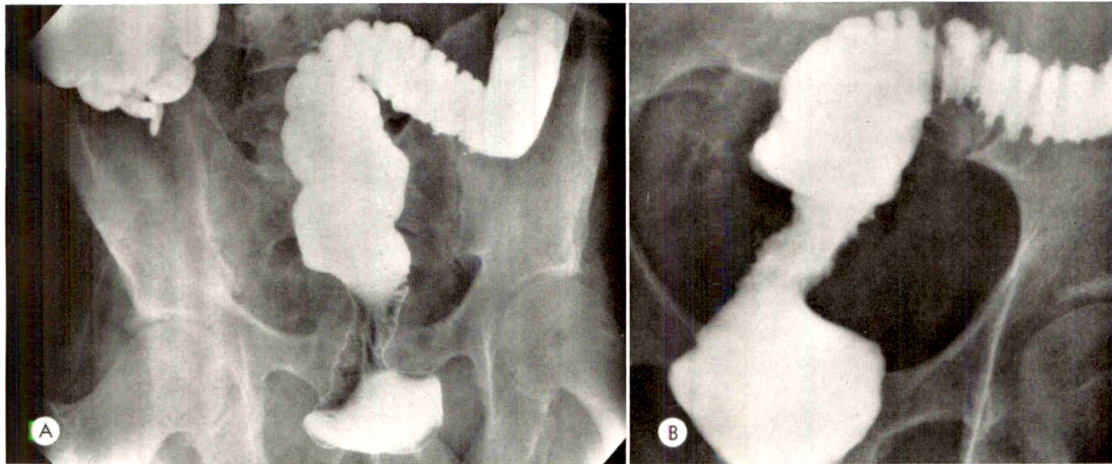


FIG. 5. Case III. (A and B) The rectosigmoid annular stricture, intact but edematous mucosa, and tapering borders of the margins of the lesion are seen. This is the most common type of colon involvement by prostatic carcinoma.

nodular prostate gland was felt. Sigmoidoscopy was unsuccessful, because of an obstruction to the passage of the sigmoidoscope at 8 cm. The mucosa seen was boggy and edematous, and there was no evidence of mucosal invasion. The rectum was rigid and fixed. The impression was that of an extraluminal lesion. The alkaline phosphatase was 5.0 King-Armstrong units and the acid phosphatase 2.5 King-Armstrong units. There were no changes in the bones on roentgen examination which could be attributed to metastatic disease. The barium enema examination showed an encircling lesion and a boggy mucosa (Fig. 5, A and B). A transurethral resection and bilateral orchiectomy were done. The histologic examination of the prostate was reported as adenocarcinoma. The patient was placed on stilbesterol and discharged from the hospital. No follow-up is available.

The combination of Type 1 and 2 is unusual. The circumferential extension appears to minimize the anterior mass, and, if the colon is surrounded by tumor, the anterior component is small. Only 1 patient in the present series had a very large anterior mass and a small circumferential component. He has been included in Type 1 in this series.

CASE IV. This 85 year old male was initially admitted to Temple University Hospital in September, 1954 with a diagnosis of benign prostatic hypertrophy. In 1959, the patient re-

turned to the hospital with symptoms of dull aching lower abdominal pain. He also complained of a recent onset of constipation. Urinary tract symptoms of frequency and nocturia, 2 to 3 times a night, and dysuria were present. On rectal examination a large hard mass on the anterior wall of the rectum was palpated which was neither mobile nor tender (Fig. 6). The clinical impression was that of an adenocarcinoma of the colon. The alkaline phosphatase was 3.6 King-Armstrong units and the acid phosphatase 12 King-Armstrong units. An intravenous urogram failed to demonstrate the right kidney. The biopsy of the mass was reported as adenocarcinoma of the prostate gland. A bilateral orchiectomy was done. The patient was placed on stilbesterol and discharged from the hospital. He was lost to follow-up until he returned to the hospital in 1962. On this admission the prostatic mass had grown, and there was marked cachexia. The admission diagnosis was carcinomatosis. The patient died 2 days after admission. At autopsy, extensive abdominal metastases were found. Both ureters were obstructed by a frozen pelvis resulting in bilateral hydronephrosis. The liver was diffusely involved with metastatic disease.

In this case, the initial colon involvement over a 4 year period extended and produced a "frozen pelvis." The barium enema study was quite atypical of primary colon carcinoma, with a large, extramucosal mass representing the prostatic malig-



FIG. 6. Case IV. A large anterior mass is combined with a small encircling component. The area of encircling is well seen in the sigmoid; it represents direct, not metastatic, extension of the prostatic carcinoma.

nancy. It is our belief that the rectal involvement was initially localized and did not represent part of the "frozen pelvis." This latter manifestation of metastatic carcinoma is a terminal stage of prostatic carcinoma, as it would be in any pelvic carcinoma.

Metastasis of primary prostatic carcinoma to the more proximal sigmoid has been reported. Winter^{11,12} lists this as one of the types of involvement of the colon. We have been impressed with the more proximal level of the involvement of the colon than one would expect, specifically in the annular stricture. Using bone landmarks, the prostate gland is located at the same level as the spines of the pelvis or the femoral heads. This places the prostate more proximal than the ampulla of the rectum. Figure 7, with the bladder filled with contrast material from intravenous urography and the colon filled with barium and air, shows the level of the prostate (especially of the large prostate) to be quite high

in relation to the rectum and actually at the rectosigmoid junction. It is logical to expect that direct involvement of the sigmoid colon should occur, since the rectosigmoid swings free of the sacrum, thus offering an easier path for colon involvement than the more distal course of extension into the presacral space. It is possible that the more proximal lesions have actually originated at the level of the prostate gland and are merely manifesting themselves in the sigmoid, rather than representing a separate metastatic lesion to the sigmoid.

CONCURRENT RECTAL AND PROSTATIC CARCINOMA

Concurrent rectal and prostatic carcinomas have been reported; Winter has also included this in his classification of rectal involvement.^{4,10,11} The possibility of concurrent lesions points out the difficulty in the occasional case of identifying the histologic origin of a carcinoma in the prostatic-rectosigmoid area. At times, exact pathologic diagnosis, especially where the tumor is very anaplastic, can be difficult. In these instances, ancillary diagnostic studies are necessary for the total evaluation of the patient. These include the serum acid phosphatase, skeletal roentgenograms for metastatic disease, and cystoscopy to evaluate



FIG. 7. The prostatic mass—as shown by its pressure effect on the bladder base—correlates with the encircling rectosigmoid lesion. Note the proximal level.

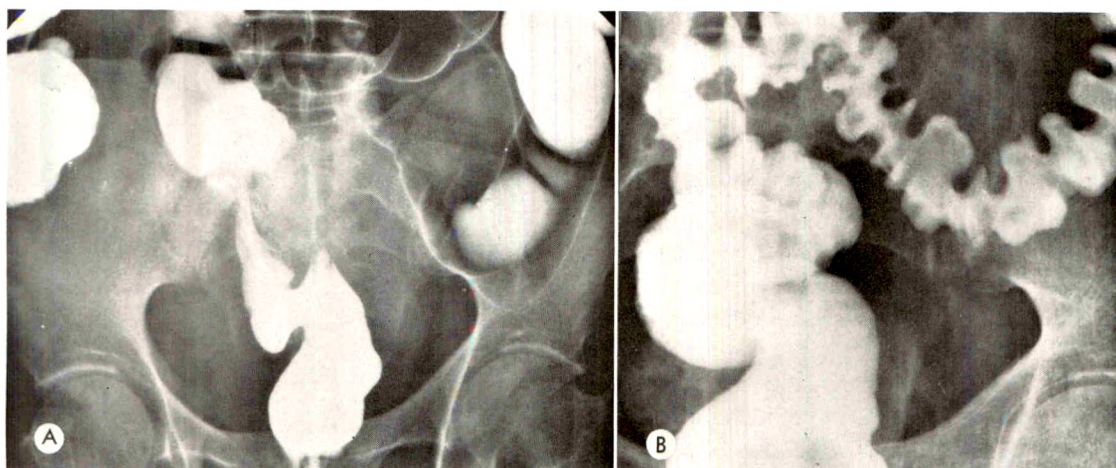


FIG. 8. Case v. (A) The typical Type B stricture is present. The mucosa is intact. The area of stricture is in the sigmoid. (B) After orchiectomy and stilbesterol, the enema examination demonstrated a normal colon. The colon involvement had disappeared.

possible bladder involvement. In the present case material, 28 per cent showed an elevated serum acid phosphatase, 28 per cent had metastatic bone disease, and in 56 per cent the cystoscopic findings were indicative of malignancy. A trial of estrogens can be the most important approach in diagnosis. Rapid reduction of tumor size and even its apparent eradication have occurred. The rapidity of change is noteworthy, reduction of tumor size in a few days having been reported.² A patient in the present case material who was treated by orchiectomy and stilbesterol had a temporary resolution of the rectal involvement.

CASE v. This 62 year old white male was admitted to Temple University Hospital in March, 1958 with the chief complaint of difficulty of urination of 7 months' duration. On rectal examination the prostate gland was reported as enlarged. The impression was that of benign prostatic hypertrophy. A barium enema study demonstrated an extrinsic pressure effect upon the sigmoid (Fig. 8A). The alkaline phosphatase was 4.1 Bodansky units and the acid phosphatase 0.98 Bodansky units. The patient had a transurethral resection and orchiectomy, and was placed on stilbesterol. He returned to the hospital in May, 1959 with the chief complaint of a poor urinary stream. The rectal examination at that time revealed a hard nodular area on the anterior rectum; however,

on barium enema study, no evidence of colon involvement was seen (Fig. 8B). The patient was discharged, but returned to the hospital in October of 1959; he died 12 days after admission with diffuse metastatic disease. An autopsy was not obtained.

In this instance, the metastatic disease was only temporarily arrested, followed by recurrence and ultimately death.

As noted in the protocols, the treatment of choice for this type of manifestation of the prostatic carcinoma is transurethral prostatic resection to relieve obstruction of the urethra, orchiectomy, and estrogen therapy. The rectal involvement represents incurable disease and radical surgery is contraindicated. The prognosis for survival is poor; it has been noted that the average patient lives 11 months after the initial visit, and the longest noted survivor lived for 27 months.³ In the present material 7 patients were available for follow-up; 2 were alive 1 and 2 years after diagnosis was made. The average survival from the time of diagnosis to death was 2 years and 10 months and the average time of survival from the colon involvement to death was 1 year and 7 months. The longest survival period was 3 years and 11 months.

Other lesions affecting the colon which should be considered in the differential diagnosis are Blumer's shelf secondary to

abdominal carcinoma,¹⁰ carcinoma of the bladder and ureter with posterior extension, and granuloma inguinale.⁶

SUMMARY

The different types of colon involvement by prostatic carcinoma have been described. In the male, the prostate gland should be suspected as the primary site, if the rectosigmoid mucosa is not affected. The diagnosis is usually readily made. In the rare instance of invasion of the mucosa, ancillary studies may be necessary for a definitive diagnosis.

The treatment of choice is hormone therapy, after a transurethral resection. However, the prognosis is unfavorable, since the rectal involvement is usually a sign of extensive disease.

In an occasional case, a dramatic regression of the carcinoma occurs with appropriate treatment. An attempt at surgical resection of the prostate and colon is not indicated.

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ROENTGENOGRAPHIC FINDINGS IN ZOLLINGER-ELLISON SYNDROME

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THE syndrome of gastric hypersecretion, atypical peptic ulceration and associated pancreatic islet-cell adenoma was first recognized by Zollinger and Ellison³¹ in 1955. Since their original report of 2 instances of this syndrome, many cases have been described, and many review articles have appeared in the literature,^{4,16} as well as reports on the pathogenesis,²³ histochemistry,^{2,3,10,18} mechanism of ulceration,¹⁰ pathologic aspects,^{2,7} clinical features,^{5,30} and surgical therapy^{1,6,8,14,32} of this condition. However, reports on roentgenographic findings in this syndrome, other than findings in individual case reports, are few.^{11,22,27} It is the purpose of this study to review roentgenographic findings of the upper part of the gastrointestinal tract in a relatively large series of cases of Zollinger-Ellison syndrome and to point out characteristic roentgenologic features that may suggest a preoperative diagnosis of the disease entity.

REVIEW OF LITERATURE

The association of atypical peptic ulceration and islet-cell adenoma of the pancreas was first reported by Forty and Barrett⁹ in 1952. Weber and associates,²⁷ in 1959, reported observations on the small bowel pattern associated with Zollinger-Ellison syndrome. They described a lace-like or cob-web appearance of the small bowel in patients with Zollinger-Ellison syndrome who presented with diarrhea as a prominent symptom. On roentgenographic examination, the small bowel showed evidence of hypersecretion and hypermotility with a thin film of contrast material outlining the valvulae conniventes resulting in a lace-like appearance. In 1960, Schlaeager and associates²² analyzed 7 cases of their

own with adenomatosis of the endocrine glands associated with atypical peptic ulceration, as well as cases from the literature, and reported on the roentgenologic features of the upper gastrointestinal tract which were characteristic of this syndrome. These roentgenographic features included evidence of ulcerations located variously throughout the duodenal loop, atonic duodenum or megaduodenum, marginal irregularities in the descending part and ulceration in the distal portion of the duodenum or upper part of the jejunum, and prominence of the mucosal folds and rigidity of the wall of the upper part of the jejunum.

Giant gastric rugae (Menetrier's disease) without peptic ulceration but with gastric hypersecretion and islet-cell adenoma of the pancreas have been known to occur.^{21,29} Association of multiple endocrine adenomas with peptic ulcer is well known,²⁵ and about 20 per cent of patients having Zollinger-Ellison syndrome have multiple endocrine adenomas.^{5,7,13,16,23,30} Ectopic islet-cell tissue in the wall of the stomach, duodenum and other extrapancreatic locations is not uncommon and has been mentioned in about 20 per cent of the reported cases.^{5,17,30}

MATERIAL AND METHODS

All cases with the diagnosis of Zollinger-Ellison syndrome at the Mayo Clinic were reviewed, but only 16 with roentgenograms of the upper part of the gastrointestinal tract were selected for study.

Criteria for selection of cases in this study were based on pathologic demonstration of islet-cell tumor associated with peptic ulceration. This category included 12 cases. Of the remaining 4, one patient had

Menetrier's disease of the stomach with islet-cell carcinoma of the pancreas without peptic ulceration; one, hyperplasia of the islet cells without tumor formation, and there were 2 cases with profuse hypersecretion and hyperacidity associated with multiple duodenal and gastrojejunal ulcerations as well as a clinical course which strongly favored a diagnosis of Zollinger-Ellison syndrome. However, during operation in one patient and operation followed by necropsy in the other, an islet-cell lesion could not be demonstrated.

Preoperative roentgenograms of the upper part of the gastrointestinal tract were available for study in 12 cases, and in 4, postsurgical roentgenograms only were available. Roentgenograms of small bowel were available in 4 cases in which diarrhea or steatorrhea was manifested during the course of illness.

RESULTS

CLINICAL FINDINGS

Eleven men and 5 women were studied. The ages of the men ranged from 36 to 60 years with an average age of 48.8 years; the ages of the women ranged from 25 to 52 years with an average age of 43.4 years at the time of diagnosis of Zollinger-Ellison syndrome. Complete clinical data, including preoperative laboratory studies, surgical treatment, and follow-up studies of these patients are reported in another paper,¹⁸ and only a few pertinent findings will be presented herein.

The time from onset of symptoms until a definitive diagnosis of Zollinger-Ellison syndrome was made ranged from 1 month to 20 years and averaged 6.3 years. Diarrhea or steatorrhea was reported as a presenting symptom in 5 cases, and was also a prominent symptom in 2 additional cases. A majority of the patients, however, had initial symptoms of epigastric distress with clinical or roentgenographic diagnosis of duodenal ulcer and a subsequent course of intractable ulcer diathesis not controlled by medical management or

any of the usually adequate surgical procedures. Sixteen patients in this series had had a total of 33 surgical procedures for peptic ulcer or complications of peptic ulcer prior to the diagnosis of Zollinger-Ellison syndrome.

PREOPERATIVE ROENTGENOGRAPHIC FINDINGS

Stomach. Analysis of preoperative roentgenograms of the upper part of the intestinal tract in 11 patients with pathologic diagnosis of Zollinger-Ellison syndrome did not reveal any uniform or characteristic changes in the stomach. Two patients had high-grade obstruction at the outlet of the stomach. In one patient having giant gastric rugae, Menetrier's disease was present, without peptic ulceration, and in another patient, evidence of prominent rugae of the stomach was seen on serial roentgenograms over a period of 5 years after the diagnosis of duodenal ulcer was made. Gastric ulceration, either alone or associated with duodenal ulcer, was not encountered in our series of cases of Zollinger-Ellison syndrome.

In one of the 2 patients having an obstructed outlet of the stomach, surgical exploration revealed a large duodenal ulcer penetrating into the head of the pancreas and a Grade 1 islet-cell carcinoma of the pancreas. In the second patient, multiple, subacute obstructing duodenal ulcers were found. Six years later, on exploration for gastrojejunal ulcer, multiple islet-cell adenomas were resected.

The report of a case of Menetrier's disease of the stomach associated with islet-cell carcinoma of the pancreas follows.

CASE 1. A 48 year old man had had epigastric distress since he was 21 years of age. For 10 years prior to his admission to the Mayo Clinic he had been on a diet for duodenal ulcer, although he had not obtained much relief of symptoms. Roentgenograms of the stomach revealed giant gastric rugae, as well as large mucosal folds in the first and second portions of the duodenum. Gastric analysis revealed fasting free acid of 100 mEq. of hydrochloric acid. Exploration and partial gastric resection

showed giant rugae of the stomach consistent with Menetrier's disease. Exploration of the pancreas revealed an encapsulated, Grade 1 islet-cell carcinoma, 2 cm. in diameter. No gastric or duodenal ulcer was found.

Prominence of gastric rugal folds was observed in 1 patient approximately 5 years after the initial diagnosis of duodenal ulcer. Roentgenograms made 9 months prior to the diagnosis of Zollinger-Ellison syndrome revealed prominent gastric rugal folds and a deficiency pattern of the small bowel. The patient had experienced the onset of diarrhea 3 months prior to roentgenographic examination of the stomach (see Case 11).

Duodenum. Roentgenographic changes in the duodenum were highly suggestive of Zollinger-Ellison syndrome. Duodenal ulceration with crater formation was seen in 4 patients, and deformity of the duodenal cap without visualization of a crater was seen in 4 additional patients. Dilatation of the duodenum distal to the deformity or ulcer crater was seen in 5 patients. Flocculation of barium in the duodenum was frequently noted in patients with Zollinger-Ellison syndrome.

The presence of a duodenal ulcer alone is not a significant finding in these patients. Location and extent of ulceration, however, may be helpful in suggesting the diagnosis. In 2 of the 4 patients, ulcer craters were demonstrated at the junction of the first and the second portion of the duodenum; in the other 2, the ulcer craters were located in the medial wall of the descending part of the duodenum (Fig. 1). In 4 patients with duodenal deformity without visualization of a crater, it was impossible to differentiate a more benign ulcer from the ulceration of Zollinger-Ellison syndrome in the absence of other features (Fig. 2).

Duodenal dilatation distal to deformity of the ulcer crater was a significant roentgenographic feature in 5 cases. The initial roentgenograms in 2 cases did not show evidence of dilatation; however, later

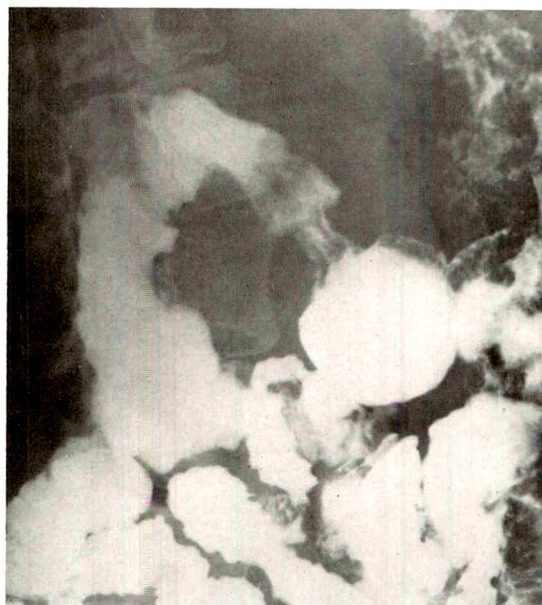


FIG. 1. Ulcer crater on medial aspect of descending portion of duodenum with dilatation distal to the ulceration. Flocculation of barium in the distal part of the duodenum and proximal part of the jejunum.

roentgenograms showed evidence of gradual development of peptic ulcer disease over 1 to 2 years (Fig. 3, *a* and *b*). The following case illustrates the development of prominent rugal folds in the stomach, as well as dilatation of the descending part of the duodenum during observation over 5 years (Fig. 4, *a*, *b* and *c*).

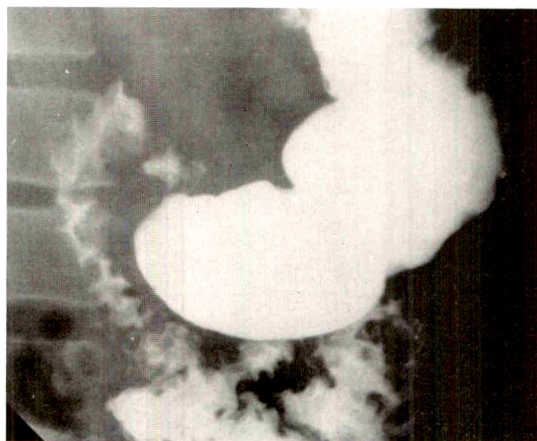


FIG. 2. Deformity of the apex of the duodenum caused by duodenal ulceration

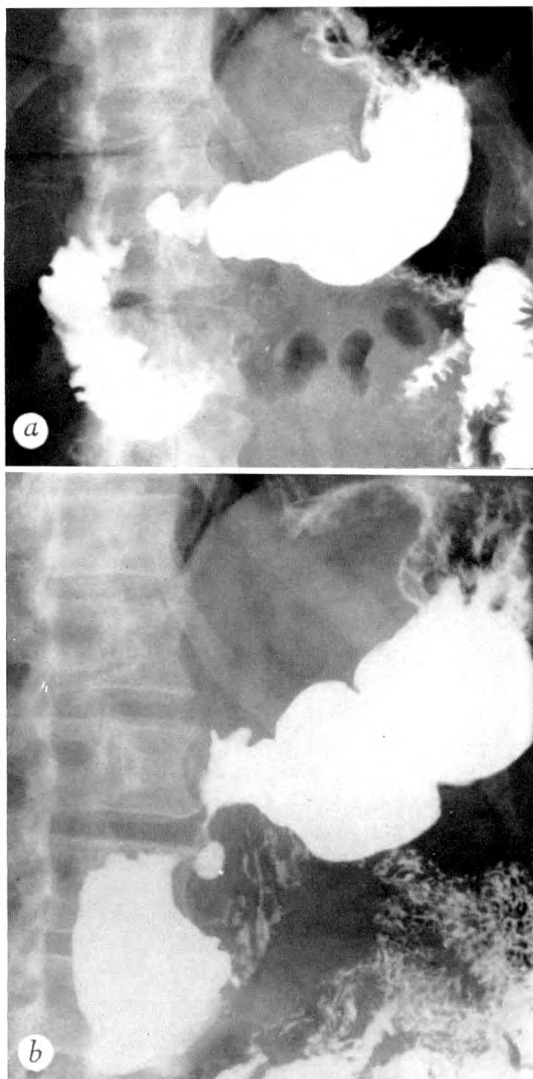


FIG. 3. (a) Deformity of the duodenal cap with ulcer crater. (b) Same region 4 years later. Severe deformity of duodenal cap with crater has developed despite medical therapy. Note the development of dilatation of the duodenum distal to the crater since last examination.

CASE II. A 60 year old man had onset of epigastric distress 20 years prior to diagnosis of Zollinger-Ellison syndrome. Roentgenologic diagnosis of duodenal ulcer was made in 1956. Exploration at this time, however, revealed chronic pancreatitis without duodenal ulceration. Cholecystectomy and choledochoduodenostomy were performed for cholelithiasis and dilatation of the common bile duct associated with edema of the head of the pancreas. Subsequent roentgenographic examinations of

the stomach revealed evidence of a deformity of the duodenum distal to the bulb as well as gradual dilatation of the distal portion of the duodenum. Roentgenograms of the stomach made in 1961 revealed evidence of prominent rugal folds and increasing dilatation of the distal portion of the duodenum. The patient had a 3 month history of diarrhea prior to the last roentgenographic examination. Roentgenograms of the small bowel revealed a deficiency pattern, and special studies revealed a pH of 1.3 in the lower descending part of the duodenum.

In January, 1962, the patient underwent subtotal gastric resection for a perforating jejunal ulcer 25 cm. beyond the ligament of Treitz. Exploration of the pancreas was not advisable. The patient died, and at necropsy multiple hemorrhagic jejunal and ileal ulcers were found. A 2 cm. ectopic islet-cell adenoma was found in the region of the splenic hilus. Gastrin-like secretagogue was extracted from the tumor.

Roentgenographic changes, other than flocculation of barium, were not observed preoperatively distal to the ligament of Treitz in our patients with Zollinger-Ellison syndrome.

POSTOPERATIVE ROENTGENOGRAPHIC FINDINGS

Gastrojejunal ulceration was reported in all 8 cases. The ulceration was extensive and involved proximal or distal loops or both.

On surgical exploration, most ulcers were found to be of the perforating type, associated with considerable jejunitis and inflammatory changes at the region of the gastrojejunostomy. Prominent gastric rugal folds, which developed 7 months after partial gastric resection and vagotomy for profuse gastric hypersecretion associated with islet-cell hyperplasia, were seen in 1 patient. This patient also had a large jejunal ulcer at the site of the gastrojejunostomy (Fig. 5, a and b).

ABSENCE OF PATHOLOGIC PROOF OF ZOLLINGER-ELLISON SYNDROME

In 2 patients an islet-cell lesion of the pancreas could not be demonstrated. Preoperative roentgenographic examination of

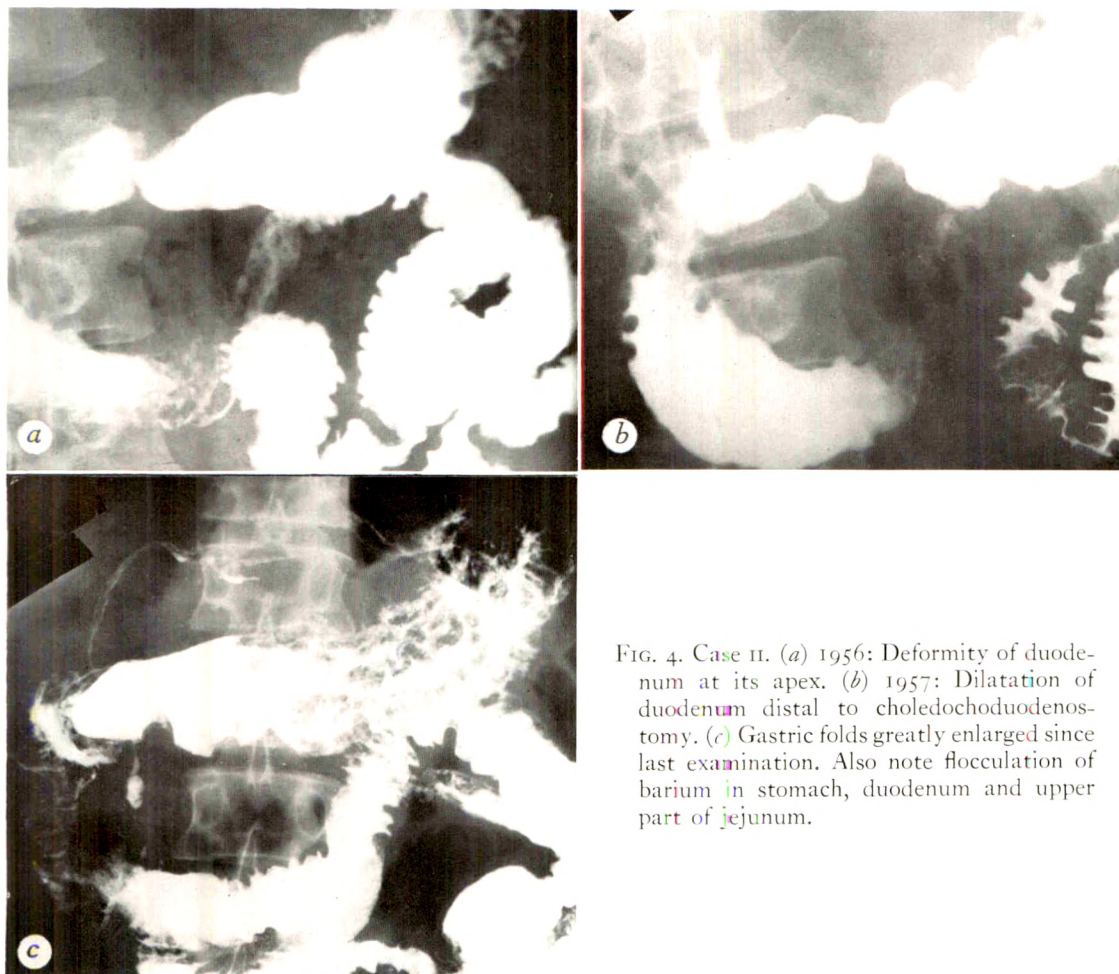


FIG. 4. Case II. (a) 1956: Deformity of duodenum at its apex. (b) 1957: Dilatation of duodenum distal to choledochoduodenostomy. (c) Gastric folds greatly enlarged since last examination. Also note flocculation of barium in stomach, duodenum and upper part of jejunum.

the stomach in 1 of these revealed prominent gastric rugae, large ulcer crater on the medial wall of the descending part of the duodenum, dilatation of the duodenum distal to the crater, and flocculation of barium in the distal portion of the duodenum and proximal portion of the jejunum (Fig. 6). The second patient underwent multiple surgical explorations for recurrent gastrojejunal ulcerations. Necropsy in the first case and partial pancreatectomy in the second did not reveal an islet-cell lesion of the pancreas.

EXAMINATION OF SMALL BOWEL

Roentgenographic examination of the small bowel was performed in 4 patients, all of whom had diarrhea or steatorrhea.

The findings were consistent with malabsorption or deficiency pattern.

PATHOLOGIC FINDINGS

Islet-cell adenoma was demonstrated in 8 patients; in 5 the adenomas were multiple. Ectopic location of islet-cell tissue was observed in 2 patients: in 1, the adenoma was located near the splenic hilus (Case II); in the other, islet-cell tissue was found in the wall of the stomach. The lesions were not recognized until after total gastrectomy.¹²

Islet-cell carcinomas of Grades I or II were found in 5 patients. Two of these patients had metastatic deposits in the liver, and another had metastatic lymph nodes near the pancreas.

Hyperplasia of islet-cell tissue was found

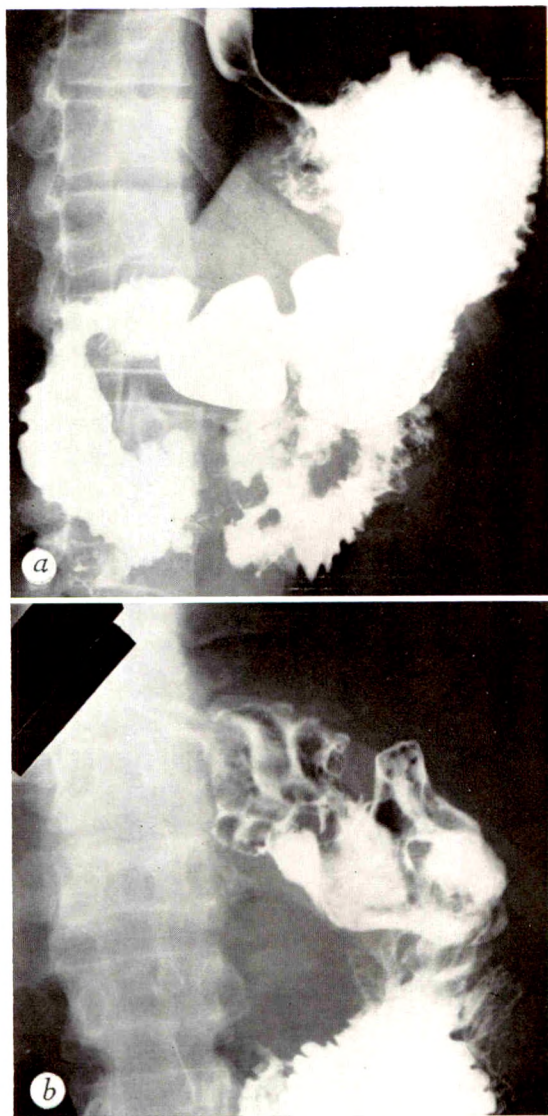


FIG. 5. (a) Dilatation of the second and third portions of the duodenum and flocculation of barium. No evidence of duodenal ulcer can be seen. (b) Eight months later, after partial gastric resection, gastric folds were greatly enlarged. Extensive jejunal ulcer developed at the site of the anastomosis.

in 1 patient, and no tumor could be demonstrated in 2 patients.

Multiple endocrine tumors and parathyroid adenomas were reported in 6 cases; in 1 case, in addition, acromegaly and enlargement of the sella were encountered and a presumptive diagnosis of pituitary adenoma had been made.

In 4 instances of adenoma and 3 of carcinoma, gastrin-like secretagogue was extracted from primary tumors, as well as from metastatic deposits in cases of carcinoma.³

COMMENT

A preoperative diagnosis of Zollinger-Ellison syndrome is seldom made.⁶ Often the presence of this disease entity is not suspected until after recurrence of peptic ulcer disease and complications such as hemorrhage and perforation following supposedly adequate surgical treatment for ulcers.

Early diagnosis and adequate therapy during initial surgical exploration are important in order to avoid multiple surgical procedures associated with increased mortality and morbidity and with complications arising after inadequate operations.^{6,20}

Absolute diagnosis of Zollinger-Ellison syndrome depends on pathologic demonstration of a pancreatic islet-cell lesion as-

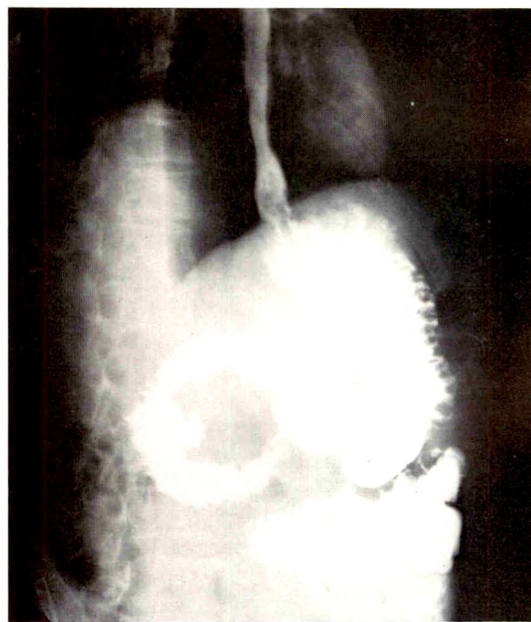


FIG. 6. Prominent rugal folds in the stomach. Large ulcer crater on the medial wall midway on the descending portion of the duodenum with some dilatation of the duodenum distally. Islet-cell adenoma could not be demonstrated in this patient.

sociated with peptic ulcer disease. However, correlation of clinical, laboratory, and roentgenographic features should, in many cases, lead to a suspicion of this disease entity, and hence to careful examination of the pancreas at the time of initial surgical exploration.

Most patients with Zollinger-Ellison syndrome present with a history of epigastric distress with little or no relief from usual ulcer regimen. Diarrhea or steatorrhea or both have been shown to occur in about a third of the patients and may precede peptic ulceration by several months to many years.^{4,7,15,19,24,26} Diarrhea was a presenting symptom in 5 of our cases and a prominent feature in 2 additional cases. The presence of other endocrine tumors, particularly parathyroid adenomas, in association with peptic ulceration, should strongly suggest Zollinger-Ellison syndrome, especially when ulcer diathesis is not controlled by parathyroidectomy. Familial incidence of endocrine adenomatosis associated with atypical peptic ulceration has been shown to occur.^{23,28}

Preoperative gastric secretory studies in patients with Zollinger-Ellison syndrome will, in most cases, reveal a high overnight 12 hour secretion (more than 1,500 ml.) and hyperacidity of well over 100 mEq. of hydrochloric acid.⁶ Histamine stimulation will not elevate appreciably the level of acidity, as these patients are probably under maximal stimulation by endogenous gastrin-like substance liberated by the islet-cell lesion. Hypokalemia and steatorrhea, with elevated fecal excretion of fat simulating relative pancreatic insufficiency, have been shown to occur in patients with diarrhea as primary manifestations of Zollinger-Ellison syndrome.^{15,19,24,26} Therefore, atypical peptic ulceration associated with diarrhea or steatorrhea should lead to suspicion of Zollinger-Ellison syndrome.

Review of preoperative roentgenograms of the upper gastrointestinal tract revealed characteristic features which, together with clinical and laboratory findings, were highly suggestive of the Zollinger-Ellison syn-

drome. Ulceration, at the apex of the duodenal cap or in its second portion, associated with dilatation of the duodenum distal to the crater or deformity was one of the most characteristic roentgenographic findings in these patients. This feature, present in 6 of 11 cases in which preoperative roentgenograms were available for study, also was seen by Schlaeger and associates²² in their patients with endocrine adenomatosis and peptic ulceration. Initial roentgenographic examination may show nonspecific ulceration. However, follow-up examination may demonstrate gradual dilatation of the descending part of the duodenum, as well as persistent duodenal ulceration even though the patient is on well-planned medical management. The reason for this dilatation is not well understood and may represent neuromuscular alteration in the wall of the duodenum associated with extreme hyperacidity. Dilatation of the proximal portion of the jejunum as reported by Schlaeger and associates was not seen in any of our cases.

Flocculation of barium in the duodenum and proximal part of the jejunum was a common finding in a majority of our cases. The appearance on the roentgenograms suggested dilution of the contrast material by excessive secretions.

An interesting observation was the development of prominent gastric rugal folds in 1 patient whose condition was followed over a period of 5 years (Case II). Prior to the last roentgenographic examination, the patient had had diarrhea for 3 months and a pH of 1.3 in the distal descending part of the duodenum. Prominent rugal folds in the stomach in asymptomatic patients, as well as in patients with more benign duodenal ulcers, are not uncommon. However, in Case II, these folds may have been the result of excessive stimulation of the parietal cell mass, as evidenced by extreme hypersecretion and low duodenal pH. Gastrin-like secretagogue was extracted from the islet-cell tumor in this case.

Demonstration of gastrojejunal ulceration on roentgenographic examination af-

ter gastric resection was one of the most frequent findings. The ulcerations usually were rather extensive, and in the majority of cases both proximal and distal loops of the jejunum were involved. The interval between gastric resection and jejunal ulceration may be several days to many years. As reported in Case II, the patient had multiple jejunal ulcers 24 hours after subtotal gastric resection, and in another case, the interval between gastric resection and postoperative jejunal ulceration was 7 years.

SUMMARY

Roentgenographic findings of the upper part of the gastrointestinal tract in patients with Zollinger-Ellison syndrome were reviewed. Preoperative roentgenograms did not reveal characteristic findings in the stomach. However, duodenal ulceration, most commonly located distal to the duodenal bulb, dilatation of the duodenum beyond the ulcerated region, and flocculation of barium in the distal portion of the duodenum and proximal portion of the jejunum were significant findings. Roentgenograms after subtotal gastrectomy revealed evidence of extensive gastrojejunal ulceration in all cases studied.

Roentgenograms of the small bowel in 4 cases did not demonstrate any characteristic changes except for findings consistent with malabsorption or deficiency state.

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ROENTGENOLOGIC-PATHOLOGIC CORRELATION OF RESECTABLE CARCINOMA OF THE PANCREATICO-DUODENAL REGION*

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THE early diagnosis of tumors of the pancreatico-duodenal region has been difficult because insufficient stress has been placed on the benefit of subtle roentgenologic findings. The pathologic correlation of these tumors with the roentgenologic changes has usually been made either on postmortem examination, when the lesion had progressed beyond reasonable limits of treatment, or on the clinical impression of the operating surgeon supported by minimal pathologic examination of the biopsy material. Any improvement in the roentgenologic diagnosis would seemingly rest on a detailed correlation between the roentgenographic findings and the anatomic-pathologic extent of the tumor.

From the therapeutic point of view, the prompt recognition of these neoplasms would facilitate radical surgical procedures, which possibly would result in a higher cure rate, this being true particularly in the non-jaundiced patient.³³

Many controversial concepts are found in the literature as to the criteria of operability of these tumors. In those cases in which a wide resection in the pancreatico-duodenal region is done, 5 year survival rates varying from 0 up to 50 per cent have been reported, depending on the clinical condition of the patient, size, location, and histologic characteristics of the lesion, absence or presence of metastatic spread and type of surgical procedure done.^{5,9,11,14,20,29,32,36} The prognosis is worse in carcinoma of the pancreas and common duct, but more encouraging in carcinomas of the papilla of Vater, duodenum, and islet cell carcinoma of the pancreas.^{20,32}

The early clinical and roentgenographic diagnosis of carcinoma of the pancreas has been extremely difficult and in many of the cases impossible. Cancer of the duodenum or the papilla of Vater, in general, is more easily detected with a careful evaluation of the duodenal loop in gastrointestinal studies with barium. Since it is difficult to differentiate the lesions mentioned above by roentgenographic means, and since the surgical approach is similar, the following discussion applies to all these tumors, as a group.

Numerous articles have been published on the subject^{3,6,8,9,15,16,17,18,22,23,24,28,31,39} and in recent years more refined techniques have been utilized in an effort to improve the accuracy and early diagnosis of these lesions.^{7,26} Many excellent reviews of the clinical and laboratory manifestations of neoplasms in the pancreatico-duodenal region have been published and will not be discussed here.^{1,10,11,12,19,32}

MATERIAL

The material consists of 29 patients with carcinoma of the pancreatico-duodenal region, operated upon at Barnes Hospital from 1943 to 1962. The following criteria were required for inclusion in the series: (1) preoperative gastrointestinal roentgenograms available for study; (2) a resectable lesion with a one-stage Whipple procedure performed; and (3) a complete pathologic examination of the resected specimen. Several patients were excluded from this study because the roentgenograms had been discarded. Carcinomas not closely related to the duodenal loop, such as

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TABLE I

ROENTGENOGRAPHIC CHANGES OF CARCINOMA OF THE PANCREATICO-DUODENAL REGION

	Pancreas		Papilla of Vater	Distal Common Duct	Duodenum
	Head	Body			
No. of Cases	12	2	12	2	1
Negative UGI*	4†	2	2	1	—
Intraluminal Mass	1	—	3	—	1
Extrinsic Pressure in 2nd Duo- denum with Intact Mucosa	6	—	5	1	—
Mucosal Ulceration in Duodenal Loop	3	—	5	—	1
Widening of Duodenal Sweep	3	—	2	—	—
Postbulbar Impression in Duodenum due to Dilated: Gallbladder	2	—	—	—	—
Common Duct	6†	—	4	1	—

* Upper gastrointestinal study.

† Two cases, negative gastrointestinal series, except for postbulbar impression due to dilated common duct.

hepatic ducts, upper common duct, and tail of the pancreas, were not included.

Roentgenograms of the stomach and duodenum outlined with barium were objectively evaluated. Special attention was given to the following findings: (1) presence of an intraluminal mass in the duodenum; (2) pressure defect in the medial wall of the descending duodenum, without alteration of the mucosa; (3) evidence of mucosal ulceration in the second portion of the duodenum; (4) widening of the duodenal loop; and (5) postbulbar indentation of the duodenum, due to dilatation of the gallbladder and/or the common duct.

After a Whipple's procedure was performed, thorough pathologic examination of the surgical specimen was done. The site of origin and characteristics of the primary lesion, invasion of surrounding structures and vessels, and spread to the lymph nodes were evaluated in each case.

The age of the patients studied varied from 36 to 73 years, the average being 58.4 years. There were 19 males and 10 females. Twenty-seven of the patients were white and 2 were Negroes; this ratio is probably related to the greater number of Caucasians admitted to this medical center.

ROENTGENOGRAPHIC FINDINGS

The retrospective study of the roentgenograms shows a higher incidence of positive findings in carcinoma of the papilla of Vater (10 cases out of 12) and less (8 cases out of 12) in carcinoma of the head of the pancreas (Table I). In the 2 cases of small neoplasms of the body of the pancreas, no demonstrable lesion could be observed in the stomach or duodenum. In 1 case of carcinoma of the distal common duct, there was evidence of extrinsic pressure in the midportion of the concave surface of the descending duodenum; the other case in this series had a normal duodenal loop. There is only 1 patient with carcinoma of the duodenum, and in this instance an area of ulceration associated with a mass was evident in the distal descending duodenum (Fig. 1, A and B).

Generally speaking, the most common finding in all the cases analyzed was an extrinsic pressure defect along the concavity of the duodenal loop, with some flattening and widening of the mucosal folds, but without gross invasion of the mucosa (Fig. 2, A-E; and 6, A-D). Some times a shallow double contour was the only abnormality in this area (Fig. 3, A-D). Interestingly enough, these were also the



FIG. 1. Carcinoma of the duodenum. Forty-two year old white female with history of tarry stools for 2 years, abdominal pain and anemia for 6 months. (A and B) There is evidence of a flat mass in the concave border of the distal descending duodenum. Destruction of the mucosal pattern and a 2 cm. in diameter ulcer crater in this area are apparent (arrow).

After a frozen section diagnosis of adenocarcinoma, a one-stage Whipple's procedure was performed. Grossly, 1 cm. distal to the papilla of Vater, there was a granular, ulcerated lesion that measured 2.5 cm. in diameter and invaded the full thickness of the duodenum. The lesion was a well differentiated adenocarcinoma. The patient is alive, without clinical evidence of local recurrence or metastasis, 6 years after the operation.

most frequently overlooked signs in the original interpretation.

Another rather constant finding was a smooth impression in the postbulbar area, due to a dilated common duct, and later-

ally, due to a distended gallbladder. However, we find it difficult to evaluate accurately this appearance, because there are a number of normal patients, without jaundice or dilatation of the common duct, who present a similar roentgenographic picture.

More easily appreciated, and not uncommon, was ulceration and irregularity in the mucosa of that portion of the duodenum invaded by the malignant process (Fig. 1, A and B; 4, A and B; and 6, A, B and C). Nevertheless, this may be overemphasized as in 2 of our cases (1 carcinoma of the pancreas and 1 of the ampulla of Vater) we felt there was destruction of the mucosa when at pathologic examination only infiltration of the submucosa was present.

An intraluminal mass was demonstrated in the 1 case of duodenal carcinoma, 1 out of 12 cases of carcinoma of the head of the pancreas and in 3 out of 12 cases of lesions in the papilla of Vater. This is a valuable point in the differential diagnosis with small tumors in the head of the pancreas, which usually do not produce an intraduodenal type of filling defect.

The presence of appreciable widening of the duodenal sweep (Fig. 5) is somewhat surprising, because we are dealing with relatively small lesions (all but 3 being less than 4 cm. in greatest dimension). This can be explained, however, on the basis of edema of the tissues surrounding the tumor and not infrequently associated pancreatitis.²⁵ Thus, the size of the mass seen on roentgenograms does not necessarily reflect the actual tumor size.

Other commonly described changes in the gastric antrum and duodenum, due to more advanced growths, such as the Frostberg sign, large filling defects and displacement of these structures, etc.,^{3,4,8,15,16,17,18,24} were not seen in our series, as extensive lesions that could not be resected by partial pancreatectomy and duodenectomy were not included.

Despite the relatively early diagnosis and apparently adequate surgical procedures, the survival rates are not very encouraging.

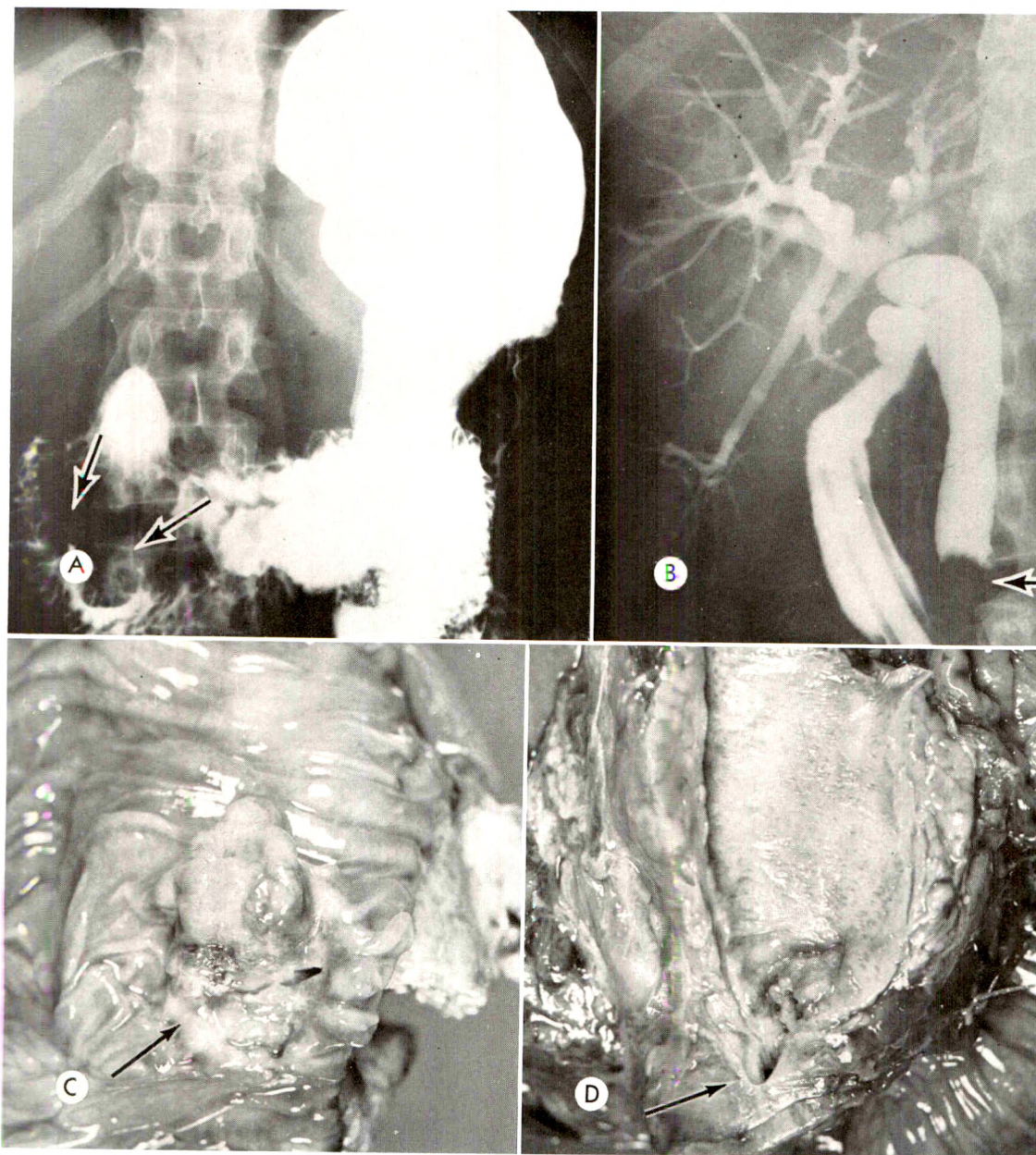


Fig. 2. Carcinoma of the ampulla of Vater. Thirty-six year old white female with weight loss, nausea and vomiting of 6 weeks' duration.

(A) A small 2.5 cm. in diameter mass is seen in the peri-ampullary region, without alteration of the mucosa. A larger extrinsic pressure defect is present in the distal second portion of the duodenum. Impression in the postbulbar region secondary to the dilatation of the common duct is apparent. (B) Cholecystocholangiogram demonstrates a filling defect producing complete obstruction in the distal common duct. There is moderate dilatation of the entire biliary tree, but more severe in the common duct.

A pancreaticoduodenectomy was done following a frozen section diagnosis of adenocarcinoma.

(C) Grossly, a polypoid tumor, 2.7 cm. in greatest dimension, replaced the papilla of Vater and extended into the head of the pancreas and the muscularis of the adjacent duodenum. A mass of ectopic pancreas was noted in the distal duodenum. (D) Opened common duct illustrating dilatation and the tumor in the distal end. Five regional lymph nodes contained metastatic carcinoma.

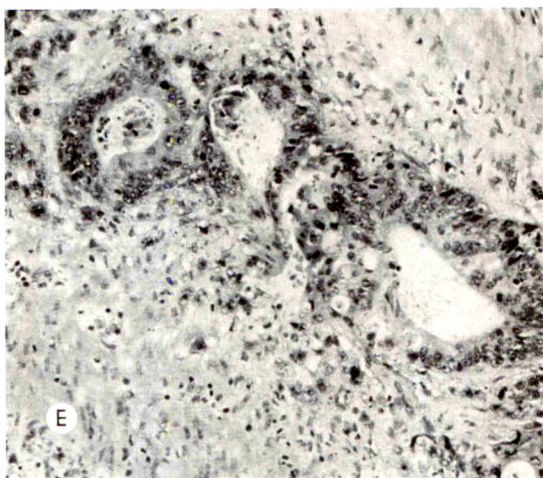


FIG. 2. (E) Histologically, the tumor was a fairly well differentiated adenocarcinoma, invasive of the duodenal muscularis (207X).

Of the 29 patients, 21 were dead within 2 years following operation, 10 of them being immediate postoperative fatalities. One patient with carcinoma of the head of the pancreas and another with ampullary carcinoma are alive, with liver and abdominal metastasis, 1 year after surgery. One other patient is alive, with no clinical evidence of local recurrence or metastasis 2 years after a Whipple's procedure was performed. The only case of carcinoma of the duodenum included in the present series is alive and well, 6 years following surgical resection. One patient operated upon for an islet cell carcinoma of the pancreatic head died 11 years later, with liver metastases proven at autopsy.

These data do not represent the results of surgical treatment of carcinoma of the pancreas and the peri-ampullary region at this institution. They apply to a selected group of patients and are cited only to emphasize the urgent need of earlier detection of these lesions. A more comprehensive study on the subject has been reported previously.²⁷

PATHOLOGY

The gross and histologic types, the patterns and the variations of carcinoma of

the pancreas and ampullary region are well known.^{25,34} All of our cases had adenocarcinoma including one islet cell carcinoma; 2 of the pancreatic carcinomas were admixed with squamous carcinoma. It was hoped that carcinomas of the areas under discussion, apparently amenable to surgical removal, would offer an opportunity to study the changes in less advanced lesions. Tabulation of the pathologic observations (Table II) show that 11 of the 14 carcinomas of the pancreas had invaded the duodenum. This is particularly striking with the knowledge that 2 of the 14 were carcinoma of the body. Five of the ampullary carcinomas invaded the duodenum locally. The pancreas was invaded 3 times. These figures are similar to an autopsy series²⁵ in which approximately 75 per cent of carcinomas of the pancreas invaded the duodenum. Our small group does not differ from the autopsy series in incidence of regional lymph node metastases. Four patients with ampullary carcinoma and 4 with pancreatic carcinoma had lymph node metastases. Our 1 case of duodenal carcinoma was confined to the duodenum, invading the muscularis locally.

Ulceration of duodenal mucosa was seen in 9 of our cases (5 ampullary carcinomas, 3 pancreatic carcinomas and 1 duodenal carcinoma). The ulcerated sites were combined with or overlying the carcinomas. Discernible masses were present in each specimen. Those of the pancreas ranged from 2 cm. to 7 cm. in greatest dimension. The tumors of the papilla were smaller, ranging from 0.5 cm. to 2 cm. in greatest dimension. Except for the duodenum, other organs were not commonly invaded by carcinoma. In only 1 case had the tumor gone beyond the duodenum, this into the transverse colon. As would be expected, in the majority the common bile duct was demonstrated to be dilated as a result of obstruction or compression by carcinoma. A common accompaniment of carcinoma of the pancreas is pancreatitis; this was seen in 10 of our cases. It was moderate to severe in degree, particularly in and near

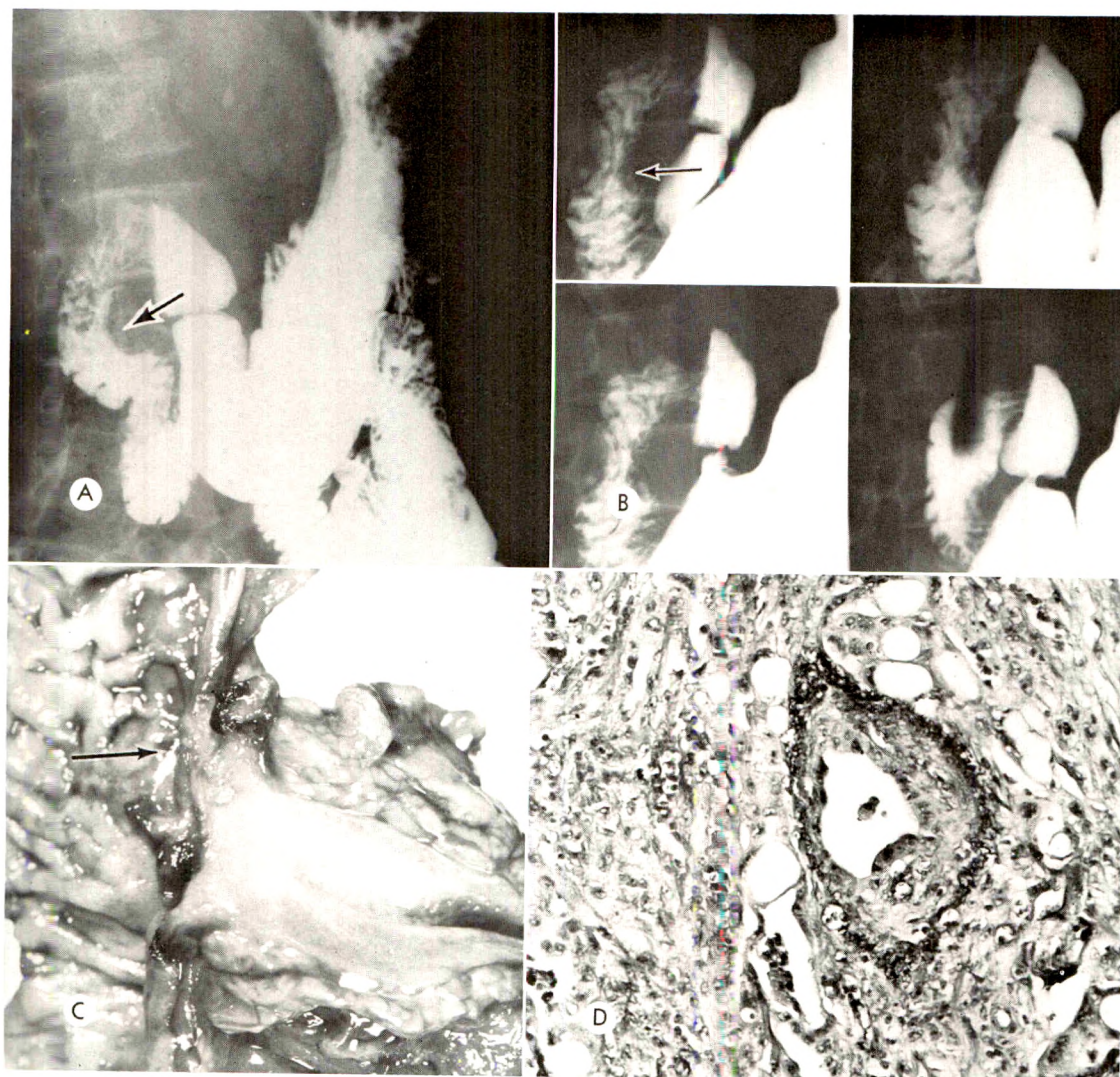


FIG. 3. Carcinoma of the peri-ampullary region. Fifty-five year old white male with a 2 month history of epigastric distress, diarrhea, weight loss, and moderate jaundice.

(A and B) There is effacement of the mucosal folds and an indentation with a double contour in the peri-ampullary region (arrows). No ulceration of the mucosa is demonstrated. Smooth pressure defects in the postbulbar area and in the convexity of the proximal duodenal loop are due to dilated common duct and gallbladder. Initially, the gastrointestinal study was felt to be within normal limits.

At abdominal exploration the gallbladder and common duct appeared considerably dilated.

(C) A 2 cm. in diameter polypoid tumor was found on the papilla of Vater, obstructing the sphincter of Oddi. There was no local tumor invasion or spread to the peripancreatic lymph nodes. (D) Microscopically, the tumor was a poorly differentiated adenocarcinoma. Here vein invasion is seen (Verhoeff-Van Gieson 262X).

the tumor bed. Fat necrosis was noted in only 1 instance.

DISCUSSION

According to a large number of reports,^{3,4,8,17,20,24,33,39} an accurate preopera-

tive roentgenographic diagnosis of lesions in the pancreatico-duodenal region is made in about 30 to 50 per cent of the cases, including both the patients with and without jaundice. However, a critical review of the upper gastrointestinal studies

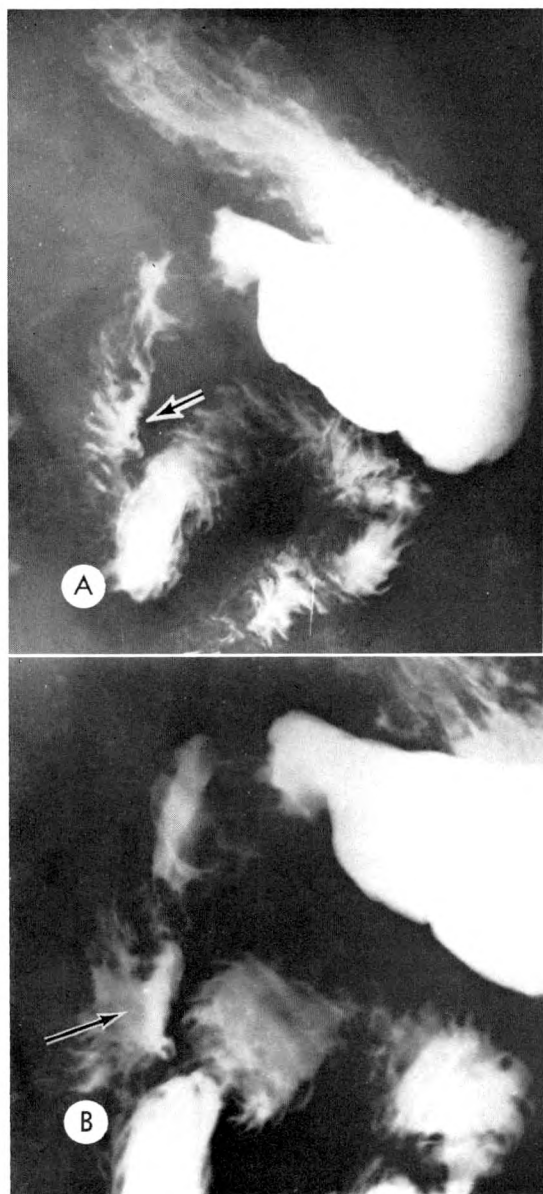


FIG. 4. Carcinoma of pancreas. Seventy year old white male with weight loss and jaundice of 2 months' duration.

(A and B) There is disruption of the mucosa with several areas of ulceration in the second portion of the duodenum.

After positive frozen section a pancreaticoduodenectomy was done. A tumor mass, $7 \times 4 \times 3$ cm., was replacing the head of the pancreas and invading through the full thickness of the duodenum and ampulla of Vater. An ulcer 1.5 cm. in diameter was present in the medial wall of the descending duodenum. There was moderate chronic pancreatitis. One of 7 regional lymph nodes contained metastatic adenocarcinoma. Microscopic diagnosis: Well differentiated adenocarcinoma.

in the current series with the "retrospectroscope," indicates that a correct roentgenographic diagnosis could have been made in approximately 70 per cent of the cases.

In the criteria of operability of these tumors, special emphasis has been given to the invasion of the portal vein and superior mesenteric artery as well as spread to the regional lymph nodes.^{25, 29, 32, 38} In our series, 1 patient had carcinomatous invasion of the portal vein, 1 carcinoma was observed in the vessels of the renal pedicle, and 2 patients had microscopic venous involvement at the tumor site. Eight out of the 29 cases had metastatic carcinoma in the regional lymph nodes. No roentgenographic manifestations of this spread could be appreciated on the roentgenograms. The size of the primary tumor is not a reliable factor

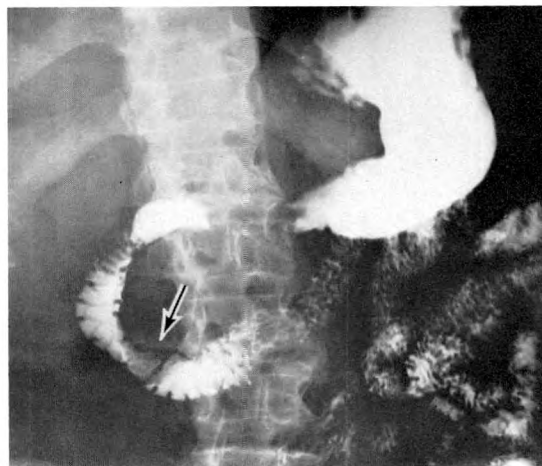


FIG. 5. Peri-ampullary carcinoma. Sixty-nine year old white male with chills, fever, and abdominal pain for 15 months and jaundice for 10 months.

The duodenal loop is widened with flattening of the mucosal folds at the medial margin. A double contour is seen distally at the junction with the third portion of the duodenum. Moderate compression deformity in the lower border of the duodenal bulb is evident as well. No intrinsic ulceration of the duodenal mucosa can be seen.

At laparotomy, the gallbladder and common duct were greatly dilated. Frozen section showed adenocarcinoma and a pancreaticoduodenectomy was performed.

Pathology: Grossly, the papilla of Vater was replaced by a friable, red mass, 1 cm. in diameter, extending into the head of the pancreas. There was associated chronic pancreatitis. Regional lymph nodes were negative for metastasis.

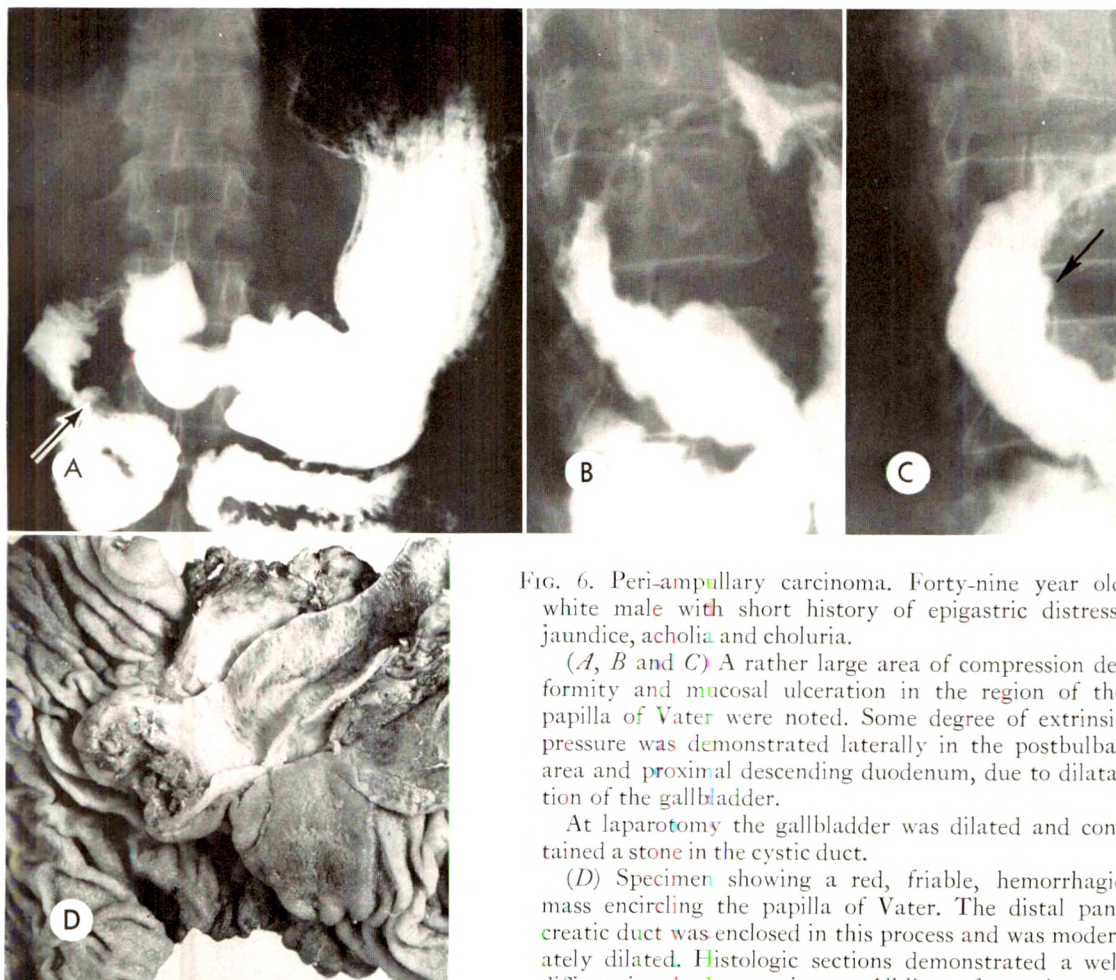


FIG. 6. Peri-ampullary carcinoma. Forty-nine year old white male with short history of epigastric distress, jaundice, acholia and choluria.

(A, B and C) A rather large area of compression deformity and mucosal ulceration in the region of the papilla of Vater were noted. Some degree of extrinsic pressure was demonstrated laterally in the postbulbar area and proximal descending duodenum, due to dilatation of the gallbladder.

At laparotomy the gallbladder was dilated and contained a stone in the cystic duct.

(D) Specimen showing a red, friable, hemorrhagic mass encircling the papilla of Vater. The distal pancreatic duct was enclosed in this process and was moderately dilated. Histologic sections demonstrated a well differentiated adenocarcinoma. All lines of resection and

regional lymph nodes were free of tumor. Chronic pancreatitis was also present. The patient is alive and well, 2 years after pancreatico-duodenectomy.

in the criteria of prognosis and operability, since we have seen small tumors with lymph node and blood vessel involvement.

It has been demonstrated that it is possible to diagnose in early stages carcinoma of the duodenum and papilla of Vater, and in a lesser number of cases carcinoma of the pancreatic head. Nevertheless, based on our roentgenographic findings and their comparison with the anatomico-pathologic features, we feel that no reliable criteria of operability have been established preoperatively. Laparotomy, meticulous inspection and palpation, and multiple frozen sections are required to better deter-

mine the full extent of the lesion. The value of biopsies in the surgical appraisal of these tumors has been indicated previously,^{2,13,35} although some surgeons have considered the procedure of limited application.^{9,30,37} It must be said that frequent consideration of small carcinomas in the pancreaticoduodenal region will lead to "overreading," but this is an unavoidable chance to be taken if early diagnosis of these lesions is to be encouraged.²¹ As reported previously by Hodes *et al.*,²¹ and others, we find it difficult in most cases, to differentiate clinically or roentgenographically pancreaticoduodenal and tumors of the papilla.

TABLE II
CARCINOMA OF THE PANCREATICO-DUODENAL REGION: PATHOLOGIC FINDINGS

	Pancreas		Papilla of Vater	Distal Common Duct	Duodenum
	Head	Body			
No. of Cases	12	2	12	2	1
Involvement of Duodenum					
Infiltration	8		5		
Ulceration	3		5		1
Lymph Node Metastases	4		4		
Distal Extension					
Liver			1		
Colon	1	1			
Vascular Invasion	4*				
Histologic Diagnosis					
Poorly Differentiated Carcinoma	5	2	3	1	
Well Differentiated Carcinoma	6		9	1	1
Islet Cell Carcinoma	1				
Dilated Common Duct	9		10	2	
Chronic Pancreatitis	10		2		
Common Duct Stones			1		
Roentgenographic Findings					
Positive	8	0	10	1	1
Negative	4	2	2	1	0

* 1 case of invasion of portal vein; 1 case of invasion of right renal pedicle.

SUMMARY

The roentgenographic manifestations of 29 cases of resected carcinoma of the pancreatiko-duodenal region are reviewed and correlated with the pathologic findings in the surgical specimens after a one-stage Whipple's procedure was performed. Retrospective study shows that a correct pre-operative diagnosis of these lesions can be achieved in about 70 per cent of the cases with adequate barium studies and detailed evaluation of the duodenal loop.

The most common findings were: (1) a smooth extrinsic pressure defect in the medial wall of the descending duodenum, with flattening of the mucosal folds. In a few instances a shallow double contour was seen; (2) deformity of the postbulbar area, due to a dilated common duct or less frequently a distended gallbladder; (3) ulceration of the duodenal mucosa, secondary to malignant infiltration; (4) intraluminal mass, more often seen in carcinoma of the papilla of Vater; and (5) chronic pancreati-

tis was present in almost all of the cases of carcinoma of the head of the pancreas. The size of the lesions was not found to be a reliable criterion of resectability or prognosis. From a pathologic standpoint, it is felt that there are some tissue and organ alterations that may contribute to roentgenographically detectable findings that would lead to an early and accurate pre-operative diagnosis of carcinoma of the pancreas, papilla of Vater and duodenum.

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IMPRESSION ON THE DUODENAL LOOP RESULTING FROM TUMOR OF THE PANCREAS*

A ROENTGENOGRAPHIC EVALUATION

By WARREN M. RUSSELL, M.D., and ALEXANDER R. MARGULIS, M.D.

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TO A large extent, detection of pancreatic tumors depends on abnormalities demonstrated in the contrast-filled duodenal loop.^{1,2,4} Tumors of the pancreas as large as 15 cm. or more are missed with disturbing frequency on routine examinations of the gastrointestinal tract. Conversely, small tumors, measuring from 2 to 4 cm., may, on occasion, be easily detected.⁴ This

apparent discrepancy is assumed to be related to the proximity of the growing tumor to the duodenal loop. A simple experiment designed to evaluate the validity of this assumption is presented.

METHOD

A. A model of the pancreas was reconstructed from a block of foam rubber. An

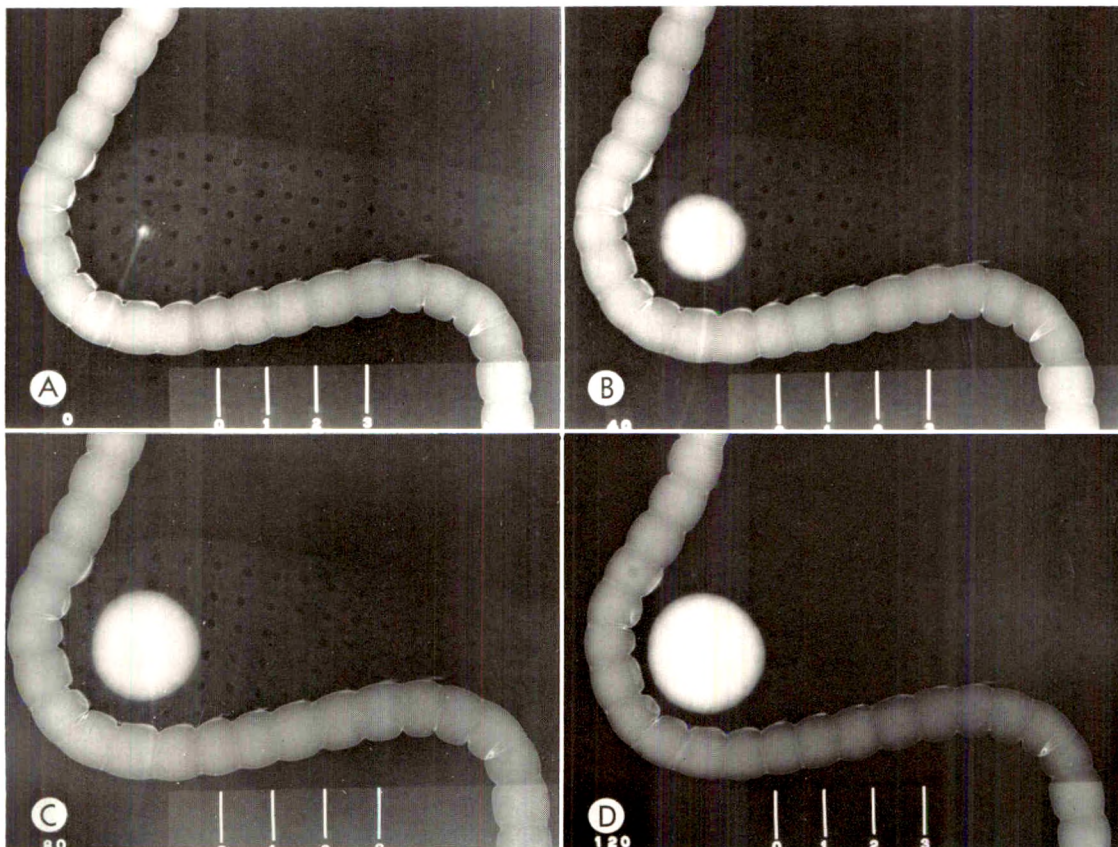


FIG. 1. (A) Foam rubber model of the pancreas with a "duodenum" of soft rubber cemented to periphery. A small balloon is in place in the center of the head of "pancreas," but is not inflated. Measurements indicate inches. (B) Balloon partially inflated with 40 ml. of water. No distortion of duodenal loop. Number in lower left refers to milliliters of water in balloon. (C) Balloon distended to 80 ml. No change. (D) Balloon fully distended with 120 ml. of water. Slight effacement of "duodenal folds."

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18 inch Penrose drain, 1 inch in diameter, was used to simulate the duodenal loop. Slightly smaller rubber bands were cemented to this rubber tube at regular intervals to simulate mucosal folds. The Penrose drain was then filled with water, but not distended, producing a soft pliable tube. This was attached to the model of the pancreas with rubber cement (Fig. 1 *A*). A small, thin-walled balloon was alternately inserted into the model at three locations: (a) at the midpoint of the "head," $1\frac{1}{2}$ inches from the "duodenal loop" (Fig. 1 *A*); (b) at the inferior margin of the head, $\frac{1}{2}$ inch from the transverse portion of the "duodenum" (Fig. 2 *A*); and (c) at the right margin of the head, $\frac{1}{2}$ inch from the descending "duodenum" (Fig. 2 *C*). In each position the balloon was distended with water to various

volumes, and roentgenograms were obtained.

B. To demonstrate the validity of these observations, the same small balloon was introduced into fresh specimens of human pancreas, still attached to the duodenal loop. The duodenum was sutured and closed at both ends and filled with a 10 per cent solution of hypaque. The balloon was distended with either water or the diluted hypaque solution, first at the center of the head of the pancreas $1\frac{1}{2}$ inches from the loop (Fig. 3 *A*) and then in the periphery $\frac{1}{2}$ inch from the descending duodenum (Fig. 4 *A*).

RESULTS

A. When the balloon was distended in the center of the reproduced pancreatic

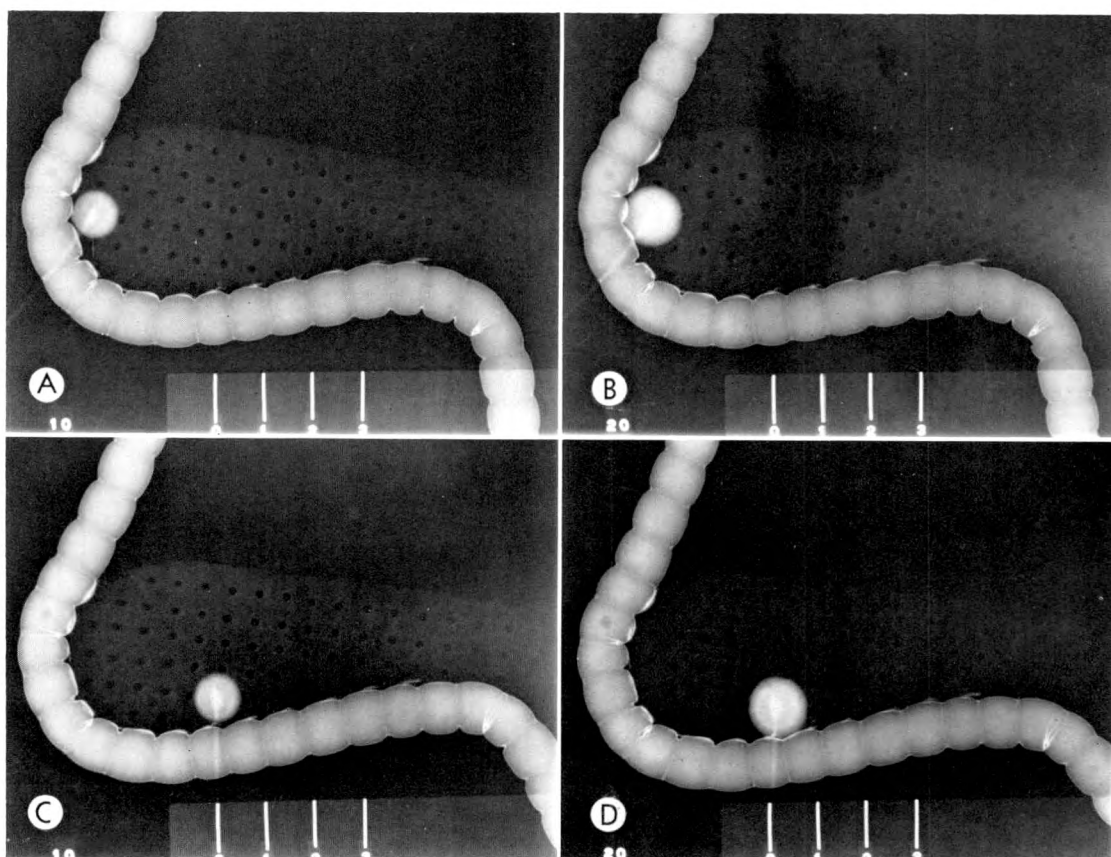


FIG. 2. (*A*) Balloon in periphery of pancreas model near loop. A small amount of distention with 10 ml. of water has resulted in slight flattening of "mucosal folds." (*B*) Balloon distended with 20 ml. of water. Definite flattening of folds. (*C*) Balloon in inferior margin of model. Distention with 10 ml. of water produces slight deformity of duodenal loop. (*D*) Distention with 20 ml. of water produces definite deformity.

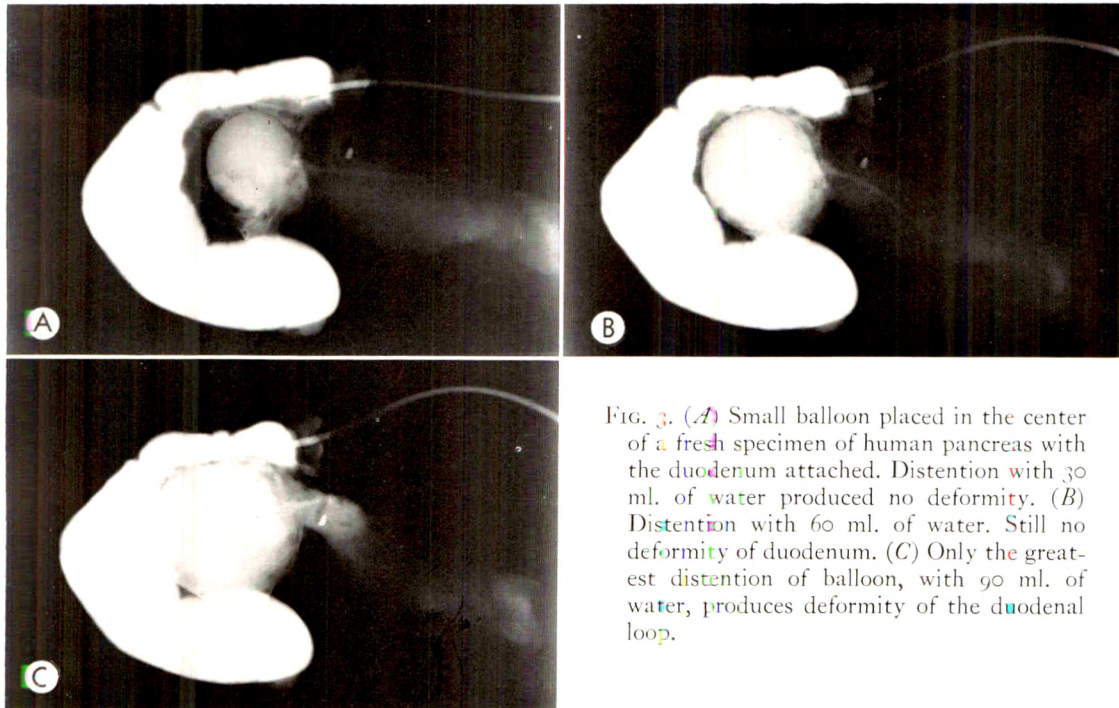


FIG. 3. (A) Small balloon placed in the center of a fresh specimen of human pancreas with the duodenum attached. Distention with 30 ml. of water produced no deformity. (B) Distention with 60 ml. of water. Still no deformity of duodenum. (C) Only the greatest distention of balloon, with 90 ml. of water, produces deformity of the duodenal loop.

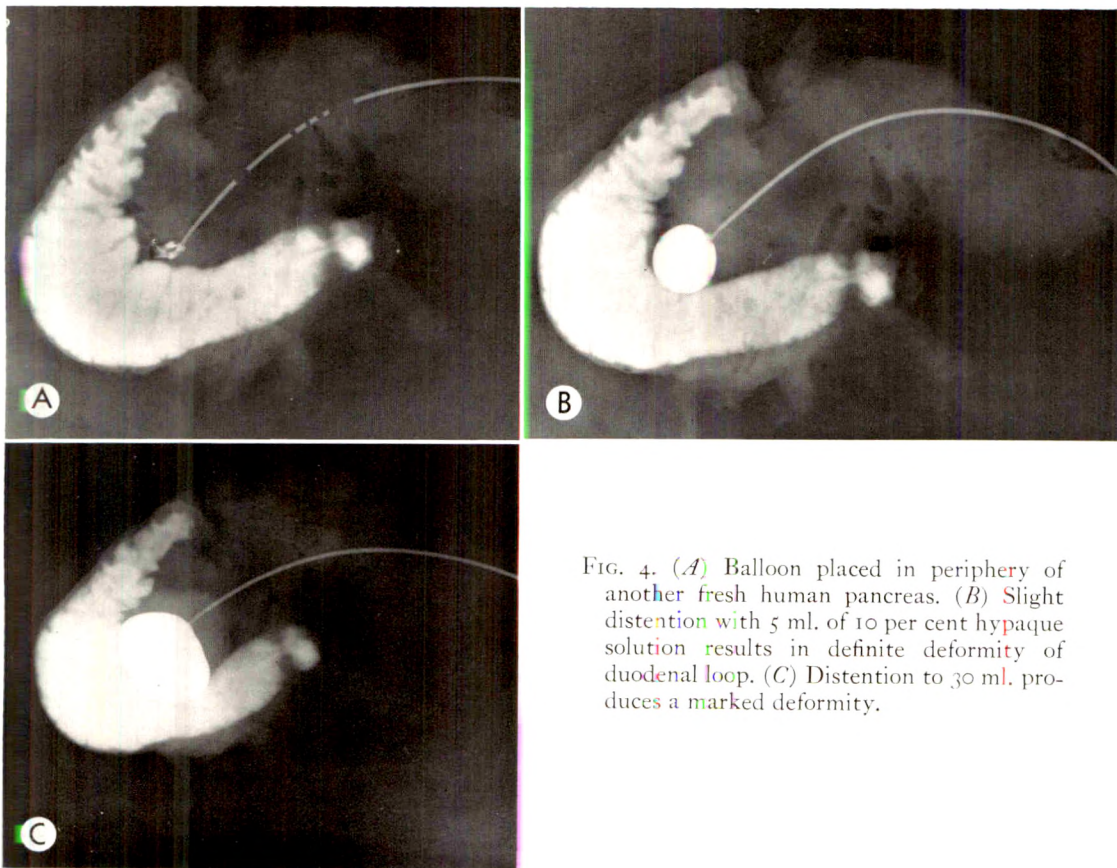


FIG. 4. (A) Balloon placed in periphery of another fresh human pancreas. (B) Slight distention with 5 ml. of 10 per cent hyaline solution results in definite deformity of duodenal loop. (C) Distention to 30 ml. produces a marked deformity.

head, slight effacement of the "mucosal folds" was produced only when the "tumor" volume reached 120 ml., a diameter of $2\frac{1}{2}$ inches (Fig. 1, *B*, *C* and *D*). With the balloon in the periphery, distention of the "tumor" to a volume of 20 ml., a diameter of $1\frac{1}{3}$ inches, caused definite deformity in the adjacent "duodenal loop" (Fig. 2, *B* and *D*).

B. Identical results were produced in the fresh specimens of human pancreas. In a central position, no deformity was produced in the contrast-filled duodenum until the volume reached 90 ml., 2 inches in diameter (Fig. 3, *B* and *C*). When the balloon was placed more peripherally, distention to only 5 ml., a diameter of $\frac{3}{4}$ inch, produced definite deformity of the duodenal loop (Fig. 4, *B* and *C*).

The effects of spasm or local invasion were not simulated in the experiment. Spasm observed in barium examinations may arouse suspicion of duodenal irritation, but may also obscure a small extrinsic deformity of the duodenal loop.

CONCLUSION

The early detection of pancreatic tumors remains a major diagnostic problem as

much today as when so described by Rigler³ in 1933. Until adequate direct means of roentgen examination of the pancreas are discovered, secondary findings in adjacent organs must provide the necessary information. This simple experiment emphasizes that location of the tumor in relation to visualized organs, particularly the duodenum, is often more significant than is absolute size. Detection of small tumors depends on a fortuitous site of origin.

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ROENTGENOLOGIC POSSIBILITIES IN SPLEEN DIAGNOSIS*

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ROENTGENOLOGIC examinations are not as yet fully utilized for the diagnosis of diseases of the spleen. The radiologist is usually asked only to determine whether surrounding organs have been affected or to ascertain the size of the spleen. However his capabilities go far beyond that. It is not only possible by a roentgenographic examination to assess the exact size of the spleen and demonstrate clinically undetectable enlargement, so-called roentgenologic splenomegaly, but, also, frequently to determine the cause and character of the enlargement.

A number of methods are at the radiologist's disposal for the examination of the spleen. Some of them yield general information while others, particularly special examinations, provide extremely precise and diagnostically unequivocal data. As a rule, several examinations must be used for an accurate diagnosis. The selection of methods is determined by the clinical situation and laboratory results, especially the hematology. The roentgenologic evaluation must be as complete as possible.

PLAIN ROENTGENOGRAPHY OF THE CHEST

The examination of the spleen should begin with a roentgenogram of the chest in the course of which special attention is paid to the left hemidiaphragm, the base of the lungs, the hilar regions and the mediastinum. In splenic disease and especially in marked splenomegaly or acute conditions, it is sometimes found that the left hemidiaphragm is elevated, deformed or restricted in movement; the left lung field may show hypoventilation with plate-like atelectasis and sometimes a small pleural effusion may be noted. In the study of the hilar regions and mediastinum, it is neces-

sary to place emphasis on enlarged lymph nodes.

PLAIN ROENTGENOGRAPHY OF THE SPLEEN

The plain roentgenogram represents the basic examination of the spleen. It should be a routine procedure and is as indispensable as the blood test. It provides information about the position, size and shape of the spleen and sometimes also about the disease processes affecting it. It also shows changes in the surrounding organs from which indirect inferences may be drawn regarding the shape of the spleen and its enlargement.

The plain roentgenogram usually shows the lower half of the spleen. Its upper part, much of which extends into the medial direction is, as a rule, covered by the fundus of the stomach. The upper pole of the spleen usually lies 2 cm. from the spinal column and may sometimes be seen in the gastric air bubble. The position of the spleen depends to a great extent on the physical constitution of the individual. In the hypersthenics, the spleen is often placed high and sometimes quite transversely. In asthenics it lies more to the side and vertically. However, respiratory movements, the filling of the surrounding organs, and the patient's position during the examination also affect the position of the spleen. In order to determine the size of the spleen, the length and width are usually measured. The length is measured from the lower pole to the highest point of the spleen; its normal value ranges between 8 and 14 cm. The width of the spleen is determined by measuring its widest transverse point rectangularly to the length; the normal values are 3 to 7 cm. Though not exact, these figures

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FIG. 1. Plain roentgenogram showing perisplenitis calcarea.

make it possible to form an approximate idea as to the size of the spleen.²⁷

If the spleen is affected by a disease process, the plain roentgenogram may show various changes in the spleen itself and secondary changes in the surrounding organs. An enlargement of the spleen is found as the most frequent abnormality; changes in position and calcification are found less often. The extent of the enlargement often affects the shape of the enlarged spleen; however, in slight splenomegaly the shape is preserved. Massive splenomegaly shows enlargement mostly in the longitudinal direction and in the lower half. Extensive focal disease processes in the spleen usually cause an irregular enlargement and an atypical form of splenomegaly. Splenic calcifications are often typical for particular diseases and from their appearance the disease process may be established.^{2,12,23}

There are certain characteristic findings in the calcifications of the wall of the splenic artery (Fig. 3, *A* and *B*) and its aneurysms, calcifications of the splenic capsule (Fig. 1) and subcapsular hematomas, sometimes also of parasitic and nonparasitic cysts, as well as in calcifications of infarcts and phleboliths of the

spleen. The appearance of splenic calcification and associated hepatic calcification with calcification of intra-abdominal lymph nodes may often lead to the diagnosis of abdominal tuberculosis. A recollection of the classic findings may help in detecting the previous administration of thorotrast (Fig. 2) or mercurial poisoning.⁸ Splenic calcification should be distinguished from calcifications in the lungs, rib cartilages, the left lobe of the liver, calcified lymph nodes, adrenals, nephrolithiasis and pancreatic lithiasis or calcification.

SUPPLEMENTARY METHODS

Insufflation of the large intestine is a valuable supplement of plain roentgenography (Fig. 4). The splenic flexure of the colon lies in a large part against the medial surface of the spleen and its filling usually outlines the spleen adequately. This examination makes it possible to ascertain the size, shape and position of the spleen and differentiates it from other enlarged soft tissue densities in the left hypochondrium. The simplicity and ease of execution together with the valuable information which may be obtained from it make insufflation of the large intestine indispensable in the diagnosis of splenomegaly. When insuffla-

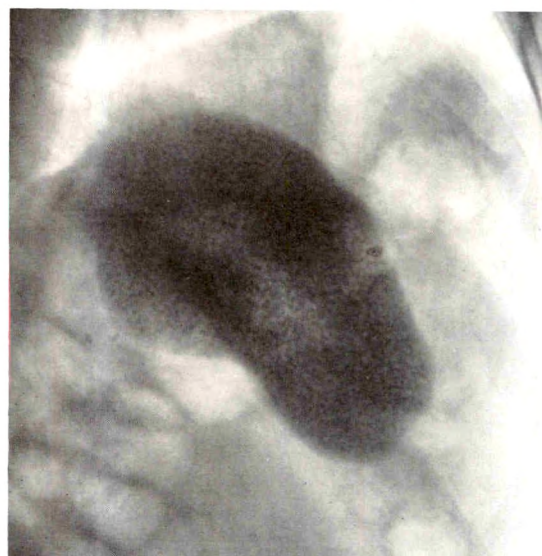


FIG. 2. Plain roentgenogram showing thorotrastosis of the spleen.

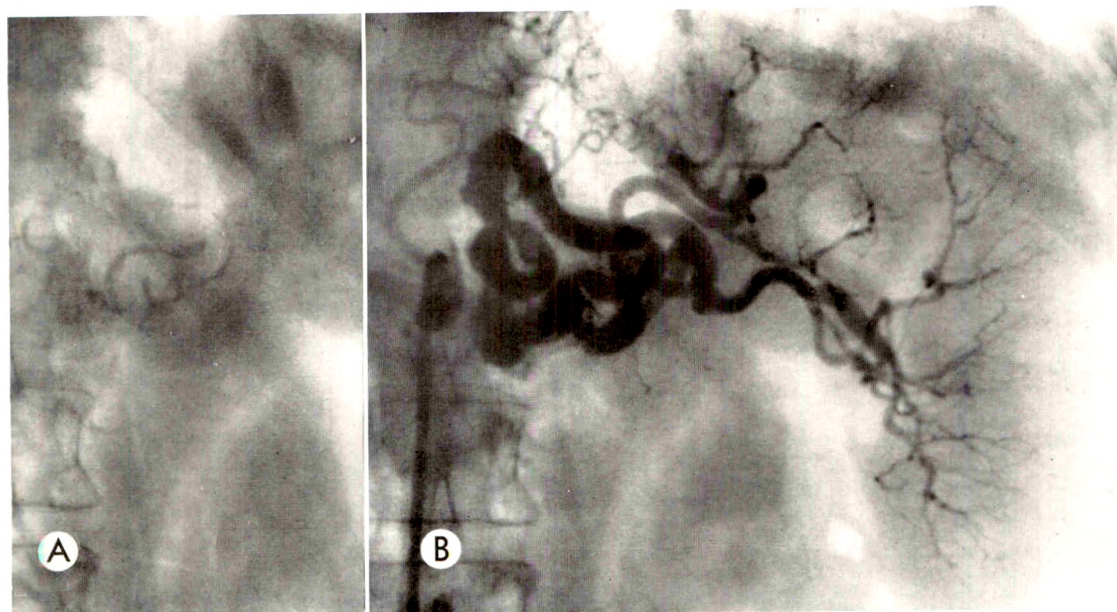


FIG. 3. (A) Plain roentgenogram showing arteriosclerosis of the splenic artery with calcifications of the wall. (B) Selective arteriogram.

tion is performed, however, the colon must not be overfilled as overdistention will cause overlapping of the spleen and difficulty in evaluation. Splenomegaly causes the contour of the hepatic flexure to alter. There is downward and medial deflection and deformity which conforms to the contour of the spleen. The contour of the splenic flexure must be considered in relation to the appearance of the rest of the large intestine.

Insufflation of the stomach also supplements the examination of the spleen and renders a more precise delineation of the shape of the spleen than the plain roentgenogram. The spleen is best visualized when the patient is in the right lateral position and horizontal beam is used. When barium is employed the spleen is best seen either in upright or supine position with the patient in the left anterior oblique position.

The gastric contour is often altered by indentation of a spleen of normal size, especially in the region of the fundus and body of the stomach (Fig. 5). Sometimes

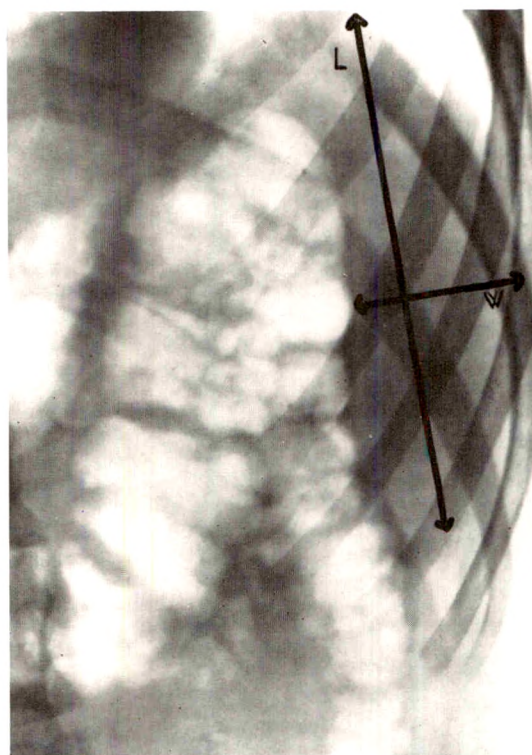


FIG. 4. Normal sized spleen with insufflation of the large intestine. L=length; W=width of the spleen.

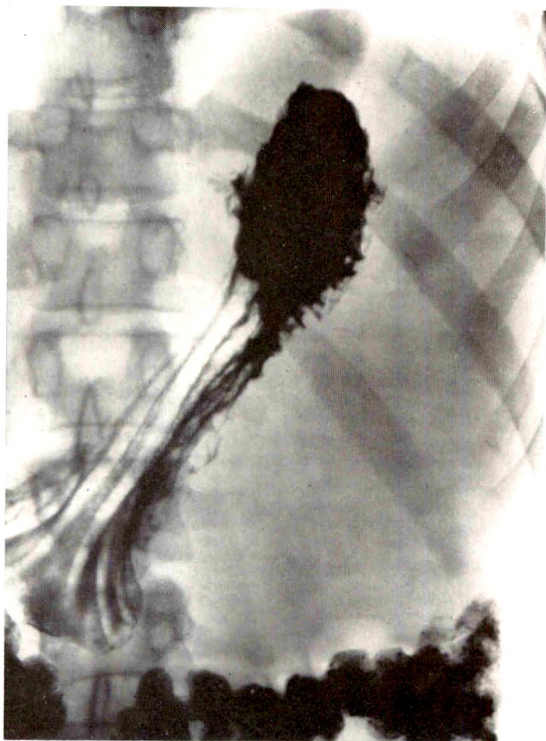


FIG. 5. Distortion of the stomach and the large intestine due to a markedly enlarged spleen.

the fundus is slightly flattened and compressed laterally and dorsally and the spleen may be wedged to a varied degree between the fundus and the hemidiaphragm. Shallow or deep impressions of the greater curvature in the region of the body of the stomach are seen, and, with enlargement of the spleen, these changes become more accentuated in the direction of enlargement. Occasionally, the fundus is completely separated from the diaphragm and, if the spleen is markedly enlarged, changes of the cardia and of the lower part of the esophagus may also be present. On the whole, the stomach is deflected medially for a varying distance. If the liver is enlarged simultaneously, pressure is exerted on the stomach from both sides causing the upper part to become narrowed. The lower part of the stomach in simultaneous hepatic and splenic enlargement is elongated and extended. The contour of the stomach in splenomegaly is generally smooth but in the case of irregularities in the splenic contour

or interposition of enlarged lymph nodes, an irregular contour is seen.

In the differential diagnosis of splenomegaly, *intravenous or retrograde pyelography* may be necessary but we have found *arteriography* to be superior to either of these (Fig. 6). Splenomegaly may cause changes in the contour and position of the left kidney. In extreme cases of splenomegaly, there may be displacement of the left kidney as far as the right half of the abdomen and enlargement of the lower pole of the spleen may cause upward displacement of the left kidney. In addition to displacement, there is usually deformity of the pelvicalyceal system of the left kidney and of the upper part of the ureter. There may be compression of renal vessels which results in functional disturbance.

Diagnostic pneumoperitoneum (Fig. 7) may be used for the precise detection of spleen morphology and especially for the differential diagnosis of the left subphrenic region. This is particularly helpful when

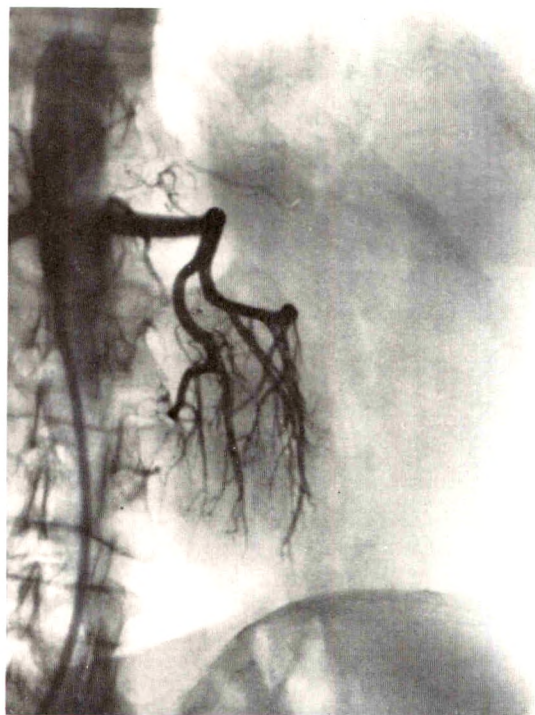


FIG. 6. Renal arteriogram showing medial dislocation of the left kidney due to a markedly enlarged spleen.

combined with insufflation of the stomach, and it reliably distinguishes primary changes in the wall of the stomach from changes caused by the spleen. It is sufficient to use 800 to 1,000 ml. of gas, preferably carbon dioxide. In pneumoperitoneum the spleen is best visualized by vertical beam if the patient is prone and by horizontal beam in the right lateral position. The splenic contour, which is well outlined by the surrounding gas, is usually smooth but there are occasional indentations on the

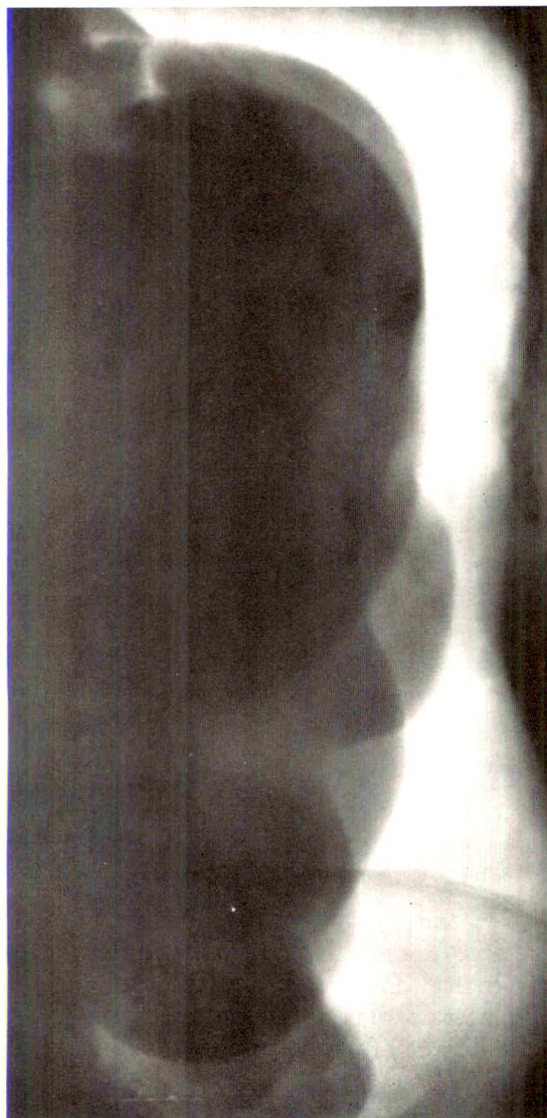


FIG. 7. Multiple metastases of reticulum cell sarcoma of the stomach into the spleen. Diagnostic pneumoperitoneum.

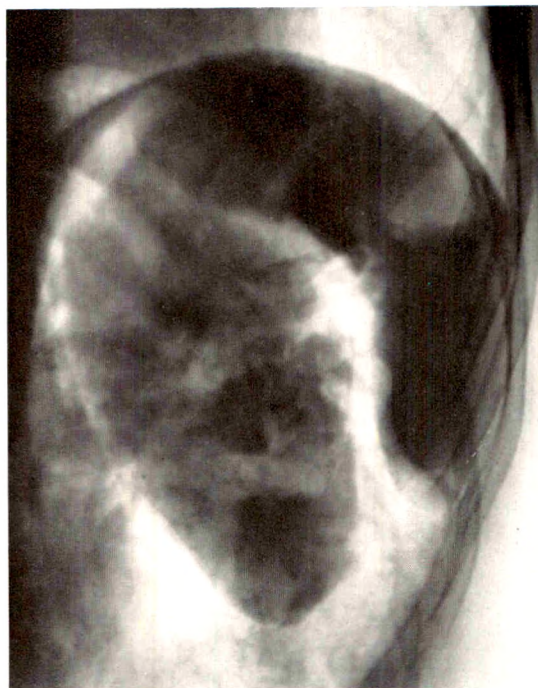


FIG. 8. Normal sized spleen in pneumoretroperitoneum.

splenic hilus. From the lower pole of the spleen around the lateral side runs the phrenicocolic ligament. The pathologic changes best shown by pneumoperitoneum are enlargement and changes in shape or outline. Protrusions and irregularities on the surface can sometimes be seen using this examination. Subcapsularly placed single foci of disease may also be detected. Adhesions are shown as slightly contrasting stripes of varying widths and in these instances the spleen will be fixed to the hemidiaphragm or other surrounding structure.

To ensure a more precise morphologic and topographic assessment of the spleen and for the purpose of differential diagnosis, especially to clarify the relationship of the spleen to the left kidney and adrenal or to tumors of the retroperitoneum, it is preferable to carry out retroperitoneal air studies (Fig. 8). When the spleen is the point of interest, the right side is the best position for insufflation with 600 to 800 ml. of gas being sufficient. Laminagrams are made in the anteroposterior projection. The spleen is seen best at a distance of from 4 to



FIG. 9. Splenoportogram showing massive splenomegaly in a blood disease. "Active portal hypertension."

11 cm. from the back, being very well separated from the kidney and the adrenal in this view. Retroperitoneal air studies show the exact size of the kidney; its length and width normally do not exceed 14 and 8 cm. respectively. To ascertain the size of the spleen in relation to its position and the physical type of the patient, it is useful to measure the distance from the upper pole of the spleen to the spinal column, which is about 2 cm. normally, and the distance of the spinal column from the outer wall of the thorax, which normally averages 13 cm.^{5,24}

SPLENOPORTOGRAPHY

Splenoportography is one of the most important special examinations. It helps in determining the cause and character of the disease of the spleen and especially the cause of enlargement. It distinguishes splenomegaly accompanying general or systemic disease from an enlargement of the spleen caused by liver disease or due to occlusion of the extrahepatic portal bed. In particular, it greatly facilitates the diagnosis of occlusion of the splenic vein or of the portal vein. Splenoportography reveals the occlusion, its location, the extent and type of the collateral circulation and sometimes aids in finding the cause of occlusion. It is the principal method by which the therapy for the occlusion and the course of surgical intervention can be determined. The study also yields very important information

about the perisplenic collateral circulation and warns of possible hemorrhage which may occur in splenectomy. Splenoportography is indicated in every case of splenomegaly where neither clinical nor any other examination could reveal its cause. In addition, it should also be carried out when the cause of the enlargement is thought to be portal thrombosis. The indications for splenoportography should be evaluated with great care in blood disease as well as in suspected focal disease of the spleen. In the latter, it is better to carry out splenic arteriography first and then resort to splenoportography, making certain that only unaffected parenchyma will be punctured during the procedure.

In splenomegaly, splenoportography (Fig. 9) shows appearances characteristic of the individual disease process. In systemic splenomegaly, there is the typical picture of "active portal hypertension" with enlargement and prolongation of vascular trunks and with normal or augmented speed of flow in the portal bed. The collateral circulatory system does not fill. Splenomegaly in liver disease is manifested mainly by changes in hepatic branches characteristic of the type and degree of the disease and may enable the precise diagnosis of the liver disease. Changes in the extrahepatic bed, especially congestion and filling of collateral vessels to a varied degree, are in this case of secondary importance. In extrahepatic portal occlusion, which is either congenital or caused by thrombosis or pressure from surrounding structures, deformation or amputation of the vein and the filling of the collateral vessels are the main features (Fig. 10). The real cause of the occlusion may sometimes be deduced from changes in the region of the affected vein. The extent of collateral circulation and the type of filled collaterals make it possible to determine the degree of congestion in the portal bed.

SPLENIC ARTERIOGRAPHY

Apart from splenoportography, splenic arteriography is a special roentgenologic examination which is indispensable for the

detection of some diseases. It clearly reveals the size, position and shape of the spleen and is the decisive examination for the diagnosis of changes in the splenic artery, focal processes in the spleen and traumatic conditions of the spleen. It reliably demonstrates sclerosis, aneurysm or thrombosis of the splenic artery as well as benign or malignant tumor, infarct or abscess of the spleen which has reached detectable size; in addition, rupture of the spleen and the type and extent of bleeding from the spleen may be recognized. The study also gives some information about the portal bed and is very important in the differential diagnosis of calcification and tumors of the left epigastrium and mid-abdomen. Selective splenic arteriography is an improvement over aortography as a method of examination. If affected by sclerosis, the splenic artery is usually elongated, tortuous and often has an irregular lumen. Aneurysms of the splenic artery may vary in character and may be solitary or multiple (Fig. 11). Their usual site of origin is at the bend of the trunk of the splenic artery or at the bifurcation of its branches. Aneurysms do not reach extensive dimensions and are not always uniformly filled because of thrombi. The splenic artery is visualized extremely well in cases of splenomegaly. Splenomegaly

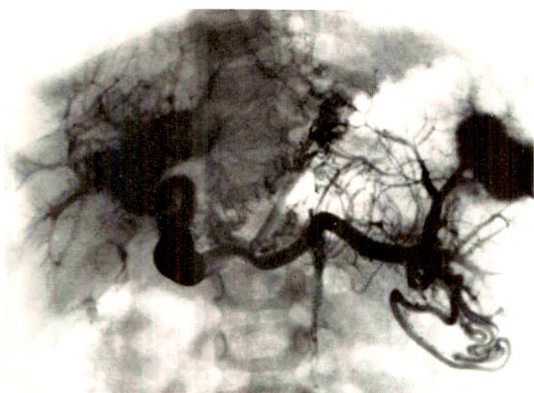


FIG. 10. Perinatally originating occlusion of the portal vein with hepatopetal collaterals in the hilus of the liver and many hepatofugal collaterals in the region of the stomach, the esophagus, the retroperitoneum and in the perisplenic area.

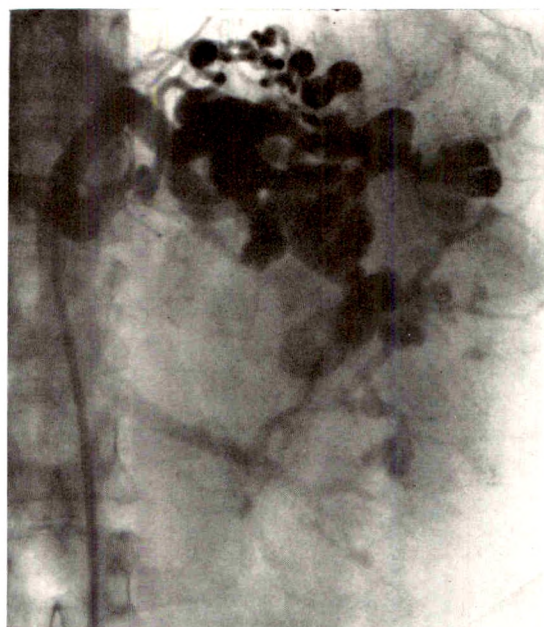


FIG. 11. Multiple congenital aneurysms of the splenic artery, its hilus and intrasplenic branches.

which is secondary to inflammatory, liver or blood disease usually shows profuse intrasplenic arterial branching with enlargement of the splenic artery itself, which has a prolonged and loop-shaped trunk. However, in splenomegaly with predominant fibrosis, the splenic artery is less enlarged and the intrasplenic branching is less profuse and distorted, the branches being for the most part narrowed and rigid. Intrasplenic branches are also less profuse and distorted in the reticuloses, while in the parenchymatous stage defects of different size appear in the splenic opacity. Focal disease processes in the spleen are demonstrated by avascular areas and by distortion of the surrounding arterial branches (Fig. 12, A and B). Splenic infarcts show avascular foci which are triangular in shape, usually with the apex of the triangle near the occluded artery. Cysts are round or ovoid and the surrounding branches appear ironed out, with atrophy of the surrounding parenchyma demonstrated by the lack of vascularity. This is shown in the parenchymatous phase of the filling as a defect in the contrast filled, unaffected parenchyma. In the case of benign tumors

with a profuse arterial component, e.g., hamartomas and sometimes also malignant tumors, there are newly formed tumor arteries with extensive networks.

Arteriography in cases of splenic trauma depends on the character of the damage. Sometimes, it is possible to see the contrast material in the peritoneal cavity and sometimes subcapsular or intraparenchymatous hematomata are manifested by avascular areas. Distortion of the surrounding splenic branches around a hematoma is often visualized and there may be contrast medium in the hematoma itself.

HISTORIC METHODS

There are reports in the literature about the *functional examination of the spleen*, which relies on the capability of the spleen to contract after the administration of certain substances. The adrenalin test has been used and, more recently, a water test has been suggested. The diagnostic possibilities of these methods are probably mini-

mal and it is felt that they do not compare with angiographic methods.

Hepatolienography has also been relegated to the background and the use of thorotrast which depicted the spleen and the liver well has been found to be toxic and has been excluded from modern diagnostic methods. At present, it is used very rarely as the method of choice in severe and life-endangering splenic thrombocytopenia, purpura or hemolytic anemia in patients who have not improved after splenectomy.¹⁵ Such patients may have a pathologic activity in an accessory spleen which may only be demonstrated by the use of thorotrast. Other contrast substances employed for hepatolienography such as iodine and fatty acid compounds, among which iodosol was best known, have also been unsuccessful because of their toxicity.

SUMMARY

The significance of roentgenologic examinations for the diagnosis of disorders of the

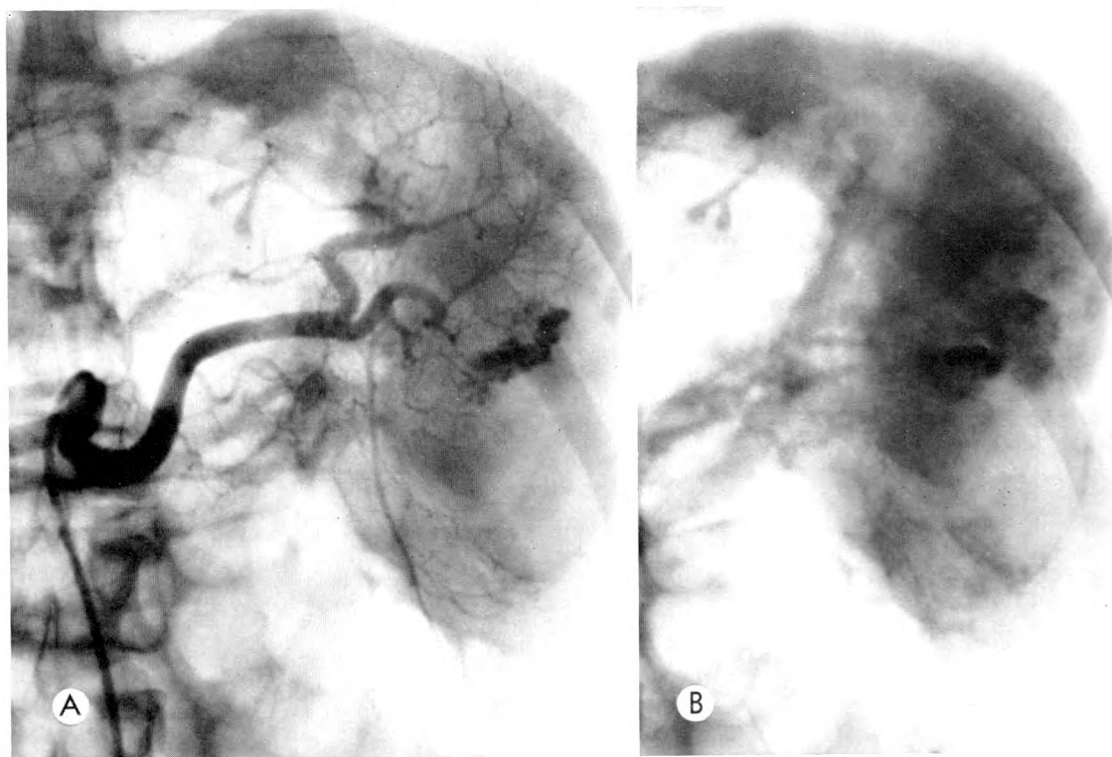


FIG. 12. Splenic arteriograms showing rupture of the spleen with subcapsular hematoma. (A) Arterial phase; (B) parenchymatous phase.

spleen is evaluated and the various methods of examination are discussed.

These methods include plain roentgenography of the spleen, chest roentgenography and examinations of the large intestine, stomach and kidney.

Pneumoperitoneum and retroperitoneal air studies with emphasis on splenoportography and arteriography are also discussed.

A brief résumé of historic methods such as the functional examination and hepatolienography is given.

The typical findings in various diseases of the spleen are presented.

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THE RELATIVE ACCURACY OF ESTIMATION OF ENLARGEMENT OF THE LIVER AND SPLEEN BY RADIOLOGIC AND CLINICAL METHODS*

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THE purpose of this study was to evaluate the relative accuracy of the determination of liver and spleen size by clinical and radiologic methods most commonly available, namely, plain radiography of the abdomen and the physical examination.

Classically, clinical enlargement of the spleen and liver has been determined by the degree of palpability below the costal margin, by the location of the palpable portion, by the area of dullness to percussion, and by the height of the diaphragm. In the majority of physical examinations, however, only the number of centimeters which the organ is palpated below the margin is recorded. This then constitutes the sole clinical criterion utilized in this study.

Radiographically, the spleen size is ordinarily related to the position of the splenic flexure and stomach, to the observed outline of the organ seen by virtue of the contrast of the fat surrounding its capsule, and to the height of the diaphragm. The size of the liver is judged by displacement of the lesser curvature of the stomach, by the position of the right flexure of the colon, and by the height of the diaphragm as well as by direct observation. All these factors are used by the radiologist in making a subjective impression of size or in any system of measurement of these organs, used to predict their size.

MATERIAL AND METHOD

This study is based on an evaluation of the records of 47 patients seen during the 5 year period, 1957 to 1961. All of these patients had at least one and usually several

clinical examinations, a roentgenogram of the abdomen within a period of 6 weeks prior to death, and a postmortem examination. A normal-sized liver was accepted as one which at postmortem examination weighed less than 2,000 gm. and a normal-sized spleen was accepted as one weighing less than 200 gm.

On the medical chart frequently several physical examinations were recorded. The examination which was carried out by the most experienced house officer was accepted for this study since this contained both positive and negative information and seemed, therefore, to be the most reliable. Clinical enlargement of the liver or spleen was accepted on the basis of palpability.

The original reports of the abdominal roentgenograms were reviewed for evaluation of liver and spleen size. If on the report there was no mention of spleen or liver, it was assumed that they had been considered to be normal in size. This constituted the subjective radiologic approach.

The roentgenograms were then reviewed without knowledge of the weight of the organs and an attempt was made to outline the margins of both liver and spleen. In all cases except one, the margin of the liver could be readily visualized. In 20 of the 47 patients, however, the shadow of the spleen could not be adequately visualized.

Several measurement systems were contrived in order to estimate mathematically the size of these organs on the basis of the abdominal roentgenograms. After the first 30 cases were examined by these several methods, the most accurate method was continued for the remainder. The final

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measurement systems used were as follows (Fig. 1): When the tip of the spleen could be identified, a vertical line was drawn from it to the left hemidiaphragm and this distance was considered to be the height of the spleen. Seven centimeters cephalad from the tip of the spleen, a line was drawn perpendicular to the height from the medial surface of the spleen to the inner border of the lateral rib cage. The spleen was then treated as a cylinder so that this measurement was halved, squared and multiplied by the height. The index figures obtained closely approximated the actual weights of the spleens (Table 1).

The spleen extends farther medially than is usually appreciated and its medial border is outlined by the perirenal fat. Most authors have accepted the medial border, which is often outlined by the air-filled transverse colon or by virtue of fat contrasting its border (Fig. 1), as the anterior edge of the spleen. (The details of the splenic shadows are to be fully described in a future publication.)

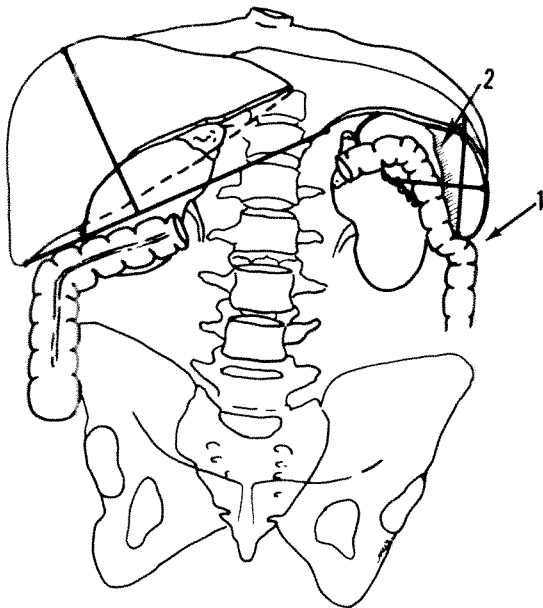


FIG. 1. Drawing showing lines used in measurement of liver and spleen volume. (1) Area of phrenicocolic ligament, related to the most inferior portion of the spleen; (2) anterior edge of spleen, related to the mesenteric portion of "splenic flexure."

The tip of the liver was identified and a line was drawn from this tip paralleling its visible lower margin to its point of estimated contact with the shadow of the diaphragm. A perpendicular was drawn from this line to the lateral portion of the right diaphragm, the exact position of this perpendicular being selected to give it maximum length. The liver was treated as a cone, the distance across the base being halved, squared and multiplied by $1\frac{1}{2}$ times the measured perpendicular.

It is apparent that many factors could be operating to invalidate this method of evaluating accuracy of determining organ size, the most serious perhaps being the 6 week interval in some cases between observation and postmortem examination, with, in other cases, significant changes occurring in the immediate premortem period. All three methods, however, have the same potential source of error and would be affected in a similar manner.

RESULTS

The results obtained are shown in Table 1.

SPLEEN

On the basis of clinical examination, 3 spleens were described as being palpable and all 3 of these were found to be enlarged at postmortem examination with weights varying from 350 to 680 gm. There was, however, no good correlation of the actual weights of these 3 spleens with the degree of palpability as described on the basis of the clinical examination. No case was found in this series, then, of a palpable spleen which was normal in size. Dell and Klinefelter² have presented evidence that this occurs and certainly many clinicians have had the experience in hypersthenic individuals of palpating a spleen, which clinically was felt not to be significant. The remaining 44 spleens were described as not being palpable. Fifteen, or approximately one-third of these, were actually larger than normal, their weights varying from 200 to 850 gm. Two additional spleens were

TABLE I

CLINICAL AND RADIOLOGIC EVALUATION OF LIVER AND SPLEEN SIZE COMPARED TO ACTUAL WEIGHT AT AUTOPSY

	Liver				Spleen			
	Clinical Evaluation	Subjective Radiology	Measure- ment	Actual Weight	Clinical Evaluation	Subjective Radiology	Measure- ment	Actual Weight
1	⊃	○	1,245	1,700	○	○	—	250
2	⊃	○	1,245	1,550	○	○	—	90
3	6 cm.	Slight ↑	2,590	2,750	8 cm.	4+	756	550
4	⊃	○	868	1,185	○	○	—	90
5	⊃	○	1,090	1,450	○	○	167	175
6	⊃	○	1,280	1,200	○	○	—	100
7	⊃	○	1,980	1,550	○	○	146	105
8	⊃	○	2,020	2,200	○	○	—	0
9	4 cm.	○	2,050	2,200	○	○	300	332
10	⊃	○	1,870	1,800	○	○	165	150
11	2 cm.	○	930	1,450	○	○	184	150
12	6 cm.	Slight ↑	1,630	2,130	2 cm.	Slight ↑	420	680
13	3 cm.	○	1,250	1,450	○	○	—	150
14	1 cm.	Marked	3,340	3,620	○	Suggestive	—	140
15	8 cm.	Some	2,480	2,900	○	○	240	440
16	8 cm.	○	1,175	1,640	○	○	—	150
17	⊃	○	1,120	1,580	○	○	194	140
18	⊃	○	1,725	2,950	○	○	—	850
19	⊃	○	—	1,500	○	○	324	375
20	⊃	○	1,200	1,300	○	○	110	175
21	⊃	○	2,300	1,700	○	○	192	175
22	⊃	○	1,350	1,400	○	○	190	100
23	8 cm.	○	1,520	1,600	○	○	230	200
24	6 cm.	Minimal	3,050	3,250	○	○	120	310
25	○	○	2,150	1,950	○	○	202	475
26	4 cm.	○	2,470	1,650	○	○	203	140
27	○	○	4,700	1,200	○	Huge	860	500
28	○	○	3,200	2,500	○	○	—	210
29	○	○	2,170	1,375	○	○	278	500
30	○	○	1,800	2,000	○	○	—	190
31	○	○	1,980	1,610	○	○	—	270
32	○	○	2,070	1,375	○	○	—	125
33	10 cm.	○	1,420	1,050	5 cm.	○	343	350
34	○	○	2,265	1,850	○	○	—	190
35	○	○	1,850	2,080	○	○	—	0
36	○	○	975	1,075	○	○	140	80
37	○	○	1,095	1,325	○	○	99	100
38	○	○	1,027	1,175	○	○	—	80
39	○	○	1,030	1,300	○	○	—	220
40	○	○	1,000	910	○	○	144	90
41	○	○	2,250	1,700	○	○	—	160
42	○	○	1,080	1,400	○	○	120	150
43	4 cm.	○	1,350	1,800	○	○	112	100
44	○	○	1,245	1,300	○	○	—	210
45	○	○	2,550	2,400	○	○	200	220
46	○	○	1,330	1,575	○	○	108	140
47	4 cm.	○	1,340	1,600	○	○	—	100

borderline in weight, leaving a conclusion that, when a spleen is described as not being palpable on clinical examination, this by no means excludes enlargement (Fig. 2 and 3).

In comparison with this, the opinion of the radiologist as recorded on the original report was examined. In 4 patients the spleen was recorded as enlarged; in 3 of these it was definitely enlarged, weighing from 500 to 680 gm. One spleen described as minimally enlarged was actually well within normal limits. In 43 cases the size of the spleen was not mentioned. In 15 of these, the spleen was definitely enlarged, weighing from 200 to 850 gm. Two spleens were borderline in size. Twenty-six of the spleens were actually normal in size. Thus, we can conclude that when enlargement of the spleen is not described on plain roentgenograms of the abdomen, splenomegaly cannot be excluded (Fig. 2 and 3).

The roentgenograms were then examined in an attempt to measure the spleen. On 20 of the studies the outline of the spleen was not clearly visible.

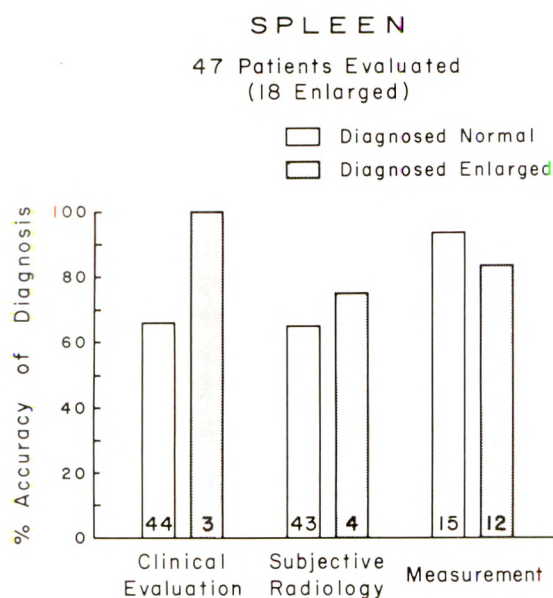


Fig. 2. Bar graph demonstrating the relative accuracy of the three methods of evaluating spleen size, broken down into the diagnosis of enlargement or normalcy.

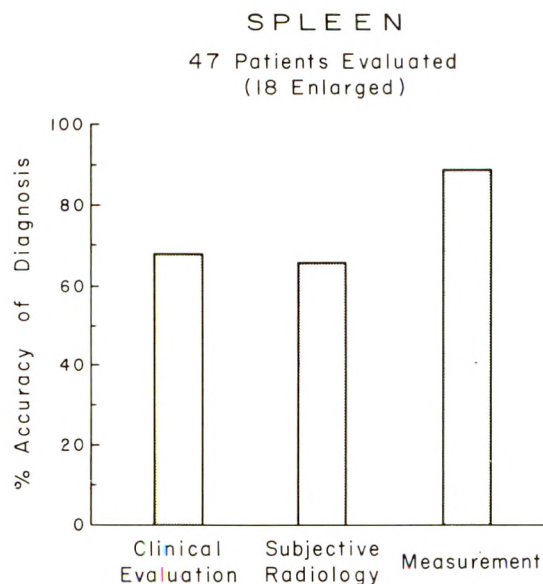


Fig. 3. Over-all accuracy of the three methods discussed in evaluating spleen size.

It is usually held that the position of the splenic flexure, as seen on the frontal roentgenogram, reflects the size of the spleen since, if the spleen is enlarged, the splenic flexure must be depressed. The displacement of the stomach to the right is considered another indication. It is, therefore, felt that if the spleen is enlarged it should be seen on a roentgenogram of the abdomen, either by virtue of its displacing these gas-filled structures or by direct visualization as a result of the fat around its capsule. Dell and Klinefelter came to this conclusion after a review of roentgenograms which, however, were centered in the area of the spleen and specifically taken for evaluation of spleen size.

Of 20 cases where the spleen could not be identified, 6 were definitely enlarged, weighing from 210 to 830 gm. and 2 more were borderline in size. With knowledge of this splenic enlargement, these roentgenograms were again reviewed, and, even in light of this knowledge, no better delineation of these enlarged spleens could be noted. It would seem, therefore, that, if the splenic shadow is not identifiable on a plain roentgenogram of the abdomen, no conclusions can be drawn as to whether the spleen is

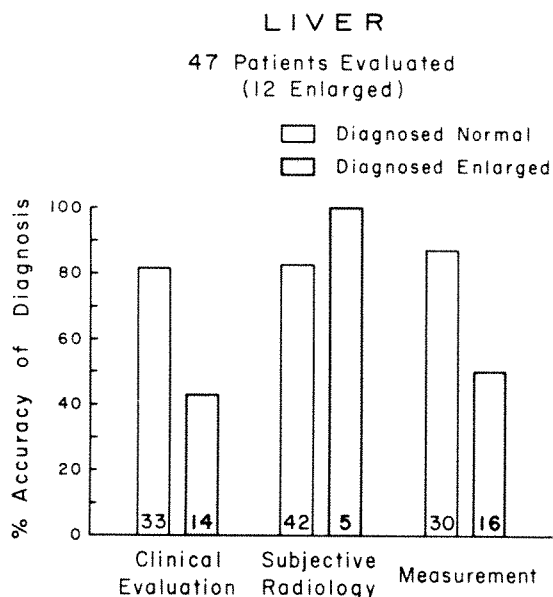


FIG. 4. Bar graph demonstrating the relative accuracy of the three methods in evaluating liver size, broken down into the diagnosis of enlargement or normalcy.

enlarged or normal in size. It also became apparent that the anatomic splenic flexure is not that portion of the colon which is radiographically called the splenic flexure, and, actually, in many cases the "radiographic splenic flexure" resides anterior to the enlarged spleen, as does the stomach. The position of the flexure, therefore, is a poor criterion for judging splenic enlargement; by using this criterion many enlarged spleens will be overlooked.

In those situations where the spleen was visible, an attempt was made to give a quantitative value to the size of spleen; the results are shown in Table 1 and Figures 2 and 3. By this method 12 spleens were measured and were noted to be definitely enlarged, and of these the weights of 10 were above the normal limit of 200 gm. This gives an 83 per cent accuracy of true enlargement in this series when the spleen was considered enlarged on the basis of this quantitative measurement. In 15 instances the spleen measured normal in size. Fourteen of these weighed within normal limits, 1 spleen weighed 310 gm., giving a 93 per

cent accuracy of normal weight when the spleen was measured as normal in size. If, however, one includes the 20 spleens which were not visualized, and considers non-visualization as a criterion for normalcy, the accuracy of this measurement decreases to approximately 69 per cent (Fig. 2 and 3).

LIVER

The clinical evaluation of the liver was similarly studied. In 14 instances the liver was described as being palpable and in only 6 of these was the liver actually enlarged by weight. The degree of extension below the costal margin seemed to bear very little or no relationship to the actual weight of the liver. Thus, palpability of the liver alone is a poor criterion for enlargement (Table 1; Fig. 4 and 5).

The remaining 33 patients were described as having a nonpalpable liver. In 6 of these, the liver was enlarged and in 1 it was borderline in size, giving a 78 per cent accuracy if one assumes that the liver is normal in size when it is not palpable (Table 1; Fig. 4 and 5).

The roentgenographic reports were analyzed for the subjective impression of the radiologist. In 5 cases the liver was de-

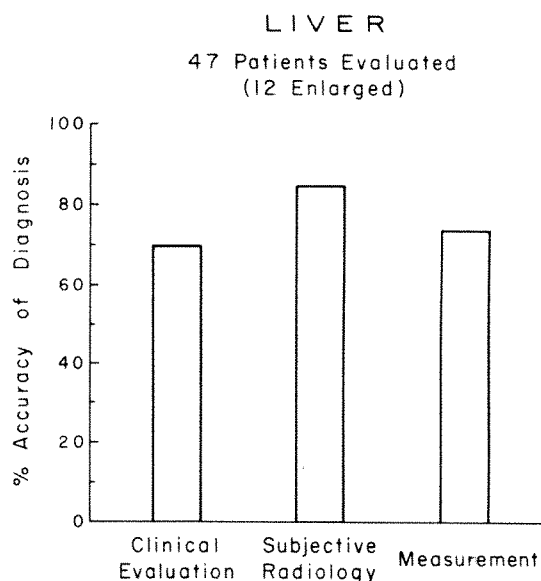


FIG. 5. Over-all accuracy of the three methods discussed in evaluating liver size.

scribed as being enlarged, and in each of these it was definitely enlarged by weight. In the remaining 42 patients, the liver was not mentioned. Seven had livers which were enlarged and 1 was borderline, giving an accuracy rate of approximately 83 per cent if one assumes that the lack of mention of liver size on roentgenograms means that the liver is normal in size (Table 1; Fig. 4 and 5).

An attempt was also made to measure the livers by using the method described in this paper. This showed 16 livers to be enlarged, of which only 8 were actually enlarged by weight, an accuracy rate of 50 per cent. Again, no good correlation was noted between the apparent degree of enlargement by measurement and the actual enlargement by weight. Of the cases, however, showing the liver to be normal by this method, 26 of 30 were actually normal in size and 2 of the remaining 4 were borderline, giving an accuracy of approximately 87 per cent (Table 1; Fig. 4 and 5).

CONCLUSIONS

An attempt was made to evaluate clinical and radiologic estimates of enlargement of the liver and spleen. For this purpose, reports of physical examinations, roentgenographic reports and plain roentgenograms of the abdomen were studied and compared. An attempt was also made to provide a system of measurement of both liver and spleen which would take into consideration the 3 dimensional geometric shape of these organs. A description of the method is given. Realizing that organ size can change immediately premortem (which change should affect all methods equally) and that the radiologic evaluation is based on plain roentgenographic examination of the abdomen, and not on roentgenograms centered over the liver and spleen, the following conclusions are drawn:

1. Palpability of the spleen probably means definite enlargement. A nonpalpable spleen, however, does not necessarily indicate a normal-sized spleen. The experience of others, that, on occasion, a palpable

spleen may be normal in size, although not verified in this study, is recognized.

2. Enlargement of the spleen as described subjectively by the radiologist is fairly good evidence of enlargement although perhaps not as accurate as definite palpability. The lack of mention of roentgenographic enlargement, however, is not a particularly good indication of the normal size of the spleen.

3. The method described for measuring the spleen proved to be most accurate both in the determination of enlarged and normal-sized spleens.

4. The lack of visualization of the spleen or the demonstration of the "radiographic splenic flexure" of the colon in apparently normal position on a frontal plain roentgenogram of the abdomen should not be interpreted as evidence that the spleen is within normal limits in size. This is not necessarily contradictory to Dell and Klinefelter's² experience since their roentgenograms were specifically made for evaluation of spleen size and, therefore, were centered over the spleen.

5. Enlargement of the liver as determined by palpability is a poor method for evaluation of hepatomegaly. Liver enlargement as ruled out by lack of palpability is somewhat more accurate. These results are probably due to the frequent failure to take into account the position of the diaphragm when assessing the significance of a palpable liver and the lack of a good method for clinically evaluating its transverse diameter.

6. The subjective description of hepatomegaly by the radiologist is a relatively accurate conclusion when used in a conservative fashion. The lack of demonstrable enlargement of the liver on roentgenograms is also relatively accurate. This appears to be more reliable than either measurement or palpability.

7. The method of measurement as described is not a particularly accurate method for evaluation of enlargement of the liver. In fact, a considerable number of normal-sized livers were judged to be en-

larged by this method. On the other hand, it does appear to be a reasonably good method for ruling out the presence of hepatomegaly.

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THE RADIOIODINATED ROSE BENGAL LIVER SCAN AS AN AID IN THE DIFFERENTIAL DIAGNOSIS OF JAUNDICE*

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THE rose bengal I¹³¹ hepatic photoscan is an examination providing not only anatomic, but also physiologic information about the liver. Because rose bengal is removed from the blood stream by the functioning polygonal cells of the liver and is excreted through the biliary tree into the intestinal tract, it supplies data not available following the use of colloidal gold which is taken up by the reticuloendothelial system, or molybdenum which enters the biliary tree in only minimal amounts. The usefulness of this test in detection of intrahepatic lesions has been previously described by others.^{3,4} Its application to clinical problems relating to jaundice will be related.

METHOD AND MATERIALS

The day following blocking of the thyroid with Lugol's solution, 1.8 μ c of radioiodinated rose bengal/kilogram of body weight is given intravenously. Twenty minutes after injection, scans are started slightly above the upper margin of the liver, to include part of the heart, and cover an area of the left upper quadrant so that radioactivity in the small bowel and/or kidneys can be depicted. If these structures are not seen on the first scan, progress scans are done at 6 and, if necessary, at 24 or more hours later. Ordinarily, one delayed scan will solve the problem of whether an obstruction is complete or partial and whether the first scan showed activity in the left kidney or the small bowel. Scan speed is adjusted to record 80 counts per centimeter. The photoscans are viewed by closed circuit television using variable

contrast. To be suitable for this viewing, the scan is made with slightly lower density than is usual so a standard x-ray illuminator provides enough light to penetrate all parts of the scan. A low contrast scan is made to avoid discarding information or emphasizing statistical variations. Only an anterior scan is done as a rule although on a few occasions a lateral scan was made to aid in identifying the kidneys.

Seventy-five hepatic photoscans of 72 patients with clinical jaundice and an elevation of the total serum bilirubin above 1.0 mg. per cent have been reviewed. The scans have been analyzed for the following features:

a. Size, shape and position of the liver and correlation with a plain roentgenogram of the abdomen.

b. Intrinsic pattern of the liver. This may be described as either uniform (normal) or patchy (abnormal). The patchy scan is due to areas of reduced activity interspersed randomly with the areas of active uptake of the isotope. A distinction from multiple masses within the liver can be made reliably.

c. Presence of discrete areas of nonfunction within the liver. Tumor, abscess, cyst and infarct may be responsible for this finding.

d. Presence of visible pattern of the biliary radicles, shown either on the early scan as a pattern of diminished activity or on a late scan by a pattern of increased activity when the active material moves from the parenchymal cells into the biliary system. Dilated ducts are more readily recognized.

* Presented at the Sixty-fifth Annual Meeting of the American Roentgen Ray Society, Minneapolis, Minnesota, September 29-October 2, 1964.

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- e. Filling of the gallbladder.
- f. Presence of the isotope in the small bowel.
- g. Presence of the isotope in the colon.
- h. Identification of the isotope in the kidneys. This occurs when there is delay in the excretion of the rose bengal by the liver.

DISCUSSION AND ILLUSTRATIVE CASES

Wagner *et al.*⁴ have pointed out the value of correlation between the scout roentgenogram of the abdomen and the hepatic scan. Whether a mass palpable in the upper abdomen is a part of the liver or simply lies adjacent to it may be resolved by a comparison of the scan with the abdominal roentgenogram.

CASE I (Fig. 1, *A* and *B*). A 65 year old white female presented with nausea, vomiting, fever and chills. Examination disclosed right upper quadrant tenderness with a palpable mass, suggesting either a greatly enlarged gallbladder or enlargement of the liver. The hepatic scan showed that the mass was deforming and compressing the parenchymal structures of the right lobe of the liver inferiorly but that the

gallbladder lay medial to the mass. There were also areas of diminished activity in the superior portion of the right lobe and just to the left of the midline in the left lobe. The diagnosis of multiple liver abscesses was made by scan and clinical findings and it was confirmed by needle aspiration. Anaerobic streptococci grew on culture of the aspirate.

The intrinsic pattern of the liver indicates whether or not there is diffuse parenchymal disease such as cirrhosis or hepatitis. On occasion, the differential diagnosis from multiple metastases may be troublesome. Christie *et al.*^{1,2} have attributed the inhomogeneous pattern in cirrhosis to low count rate, high background cut-off and/or low dot factor. While the patchy pattern can be produced by such a technique, the method described above has avoided this pitfall.

CASE II (Fig. 2). A 44 year old jaundiced white female presented with severe upper abdominal pain of 4 days' duration. The total bilirubin was 19.2 mg. per cent. The rose bengal hepatic photoscan showed an inhomogeneous pattern throughout the liver and activity in the

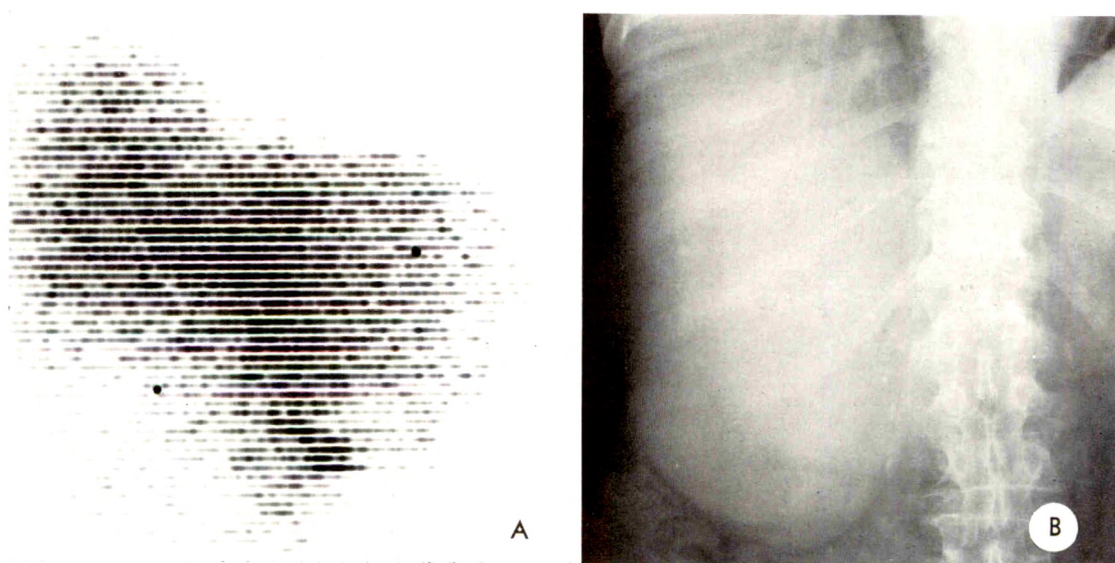


FIG. 1. Case I. Multiple hepatic abscesses, the largest in the lower portion of the right lobe. (*A*) January 17, 1964. Anterior rose bengal hepatic photoscan. The liver is enlarged. There is a large defect in the lower portion of the right lobe and smaller areas of diminished activity in the upper portion of the right lobe and in the left lobe. The gallbladder is seen just medial to the large defect. (*B*) A roentgenogram of the right upper abdomen shows a large mass in the region of the right lobe of the liver which by hepatic scan is shown to be within the liver.

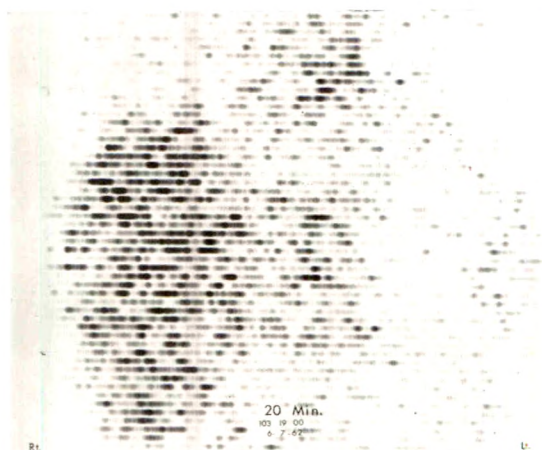


FIG. 2. Case II. June 7, 1962. Anterior rose bengal hepatic photoscan of a patient with infectious hepatitis. The photoscan shows an inhomogeneous or patchy uptake throughout the liver. There is good depiction of the heart in the upper midline portion of the scan. There is evidence of some activity in the region of the upper small bowel in the patient's left upper quadrant.

heart. There was no evidence of obstruction as the isotope was seen in the upper gastrointestinal tract on the 20 minute scan. This was a case of infectious hepatitis.

The appearance of circumscribed areas of diminished activity in the liver scans of

jaundiced patients may be due to a variety of causes including neoplasm, abscess, cyst and infarct. Infarction of the liver is illustrated in Case III.

CASE III (Fig. 3, A-D). The patient was a 49 year old white female who entered the hospital because of progressive swelling of the abdomen for 3 weeks. Physical examination disclosed jaundice, abdominal swelling and edema of the lower extremities. The rose bengal hepatic photoscan showed large portions of the right side of the liver to be nonfunctioning. Subsequently, an inferior venacavagram showed thrombi in the inferior vena cava extending to the right atrium. At autopsy, the diagnosis of Budd-Chiari syndrome was established. Thrombosis of the left hepatic vein and multiple thrombi in small veins were demonstrated. Frank infarction was found in areas of the liver.

Occasionally, portions of a normal sized biliary tree can be identified, but more often it is seen when enlarged by obstruction. Ducts are more readily seen when "hot" than when "cold." In 45 per cent of extrahepatic biliary obstruction cases, the duct system could be seen, as compared to only 24 per cent in diffuse parenchymal disease.

CASE IV (Fig. 4, A and B). A 60 year old

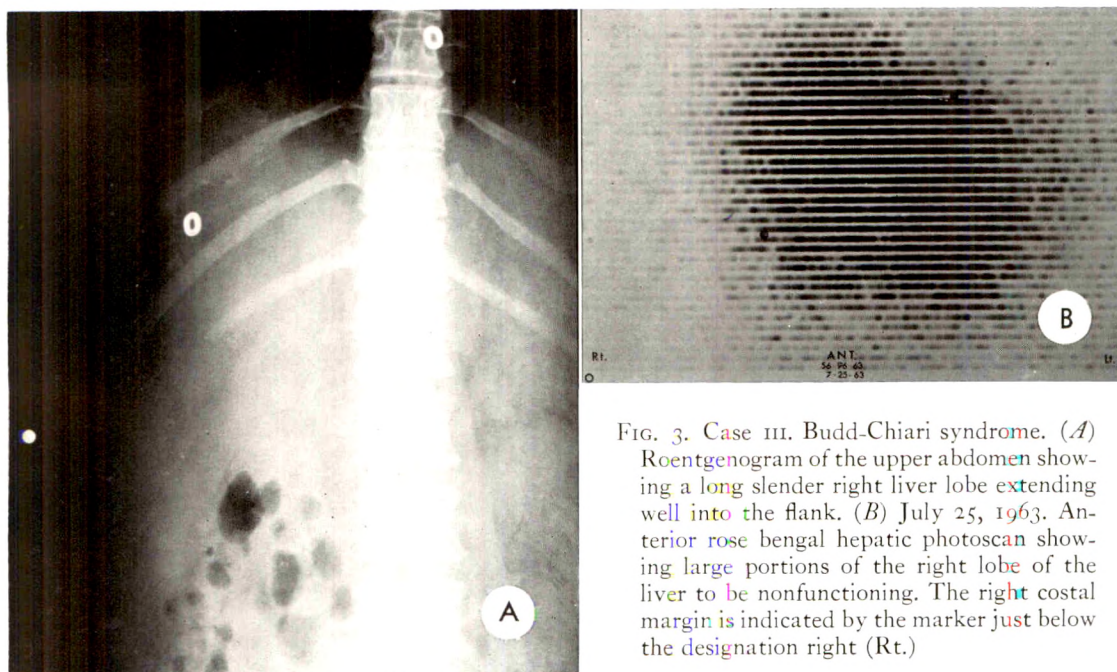


FIG. 3. Case III. Budd-Chiari syndrome. (A) Roentgenogram of the upper abdomen showing a long slender right liver lobe extending well into the flank. (B) July 25, 1963. Anterior rose bengal hepatic photoscan showing large portions of the right lobe of the liver to be nonfunctioning. The right costal margin is indicated by the marker just below the designation right (Rt.)

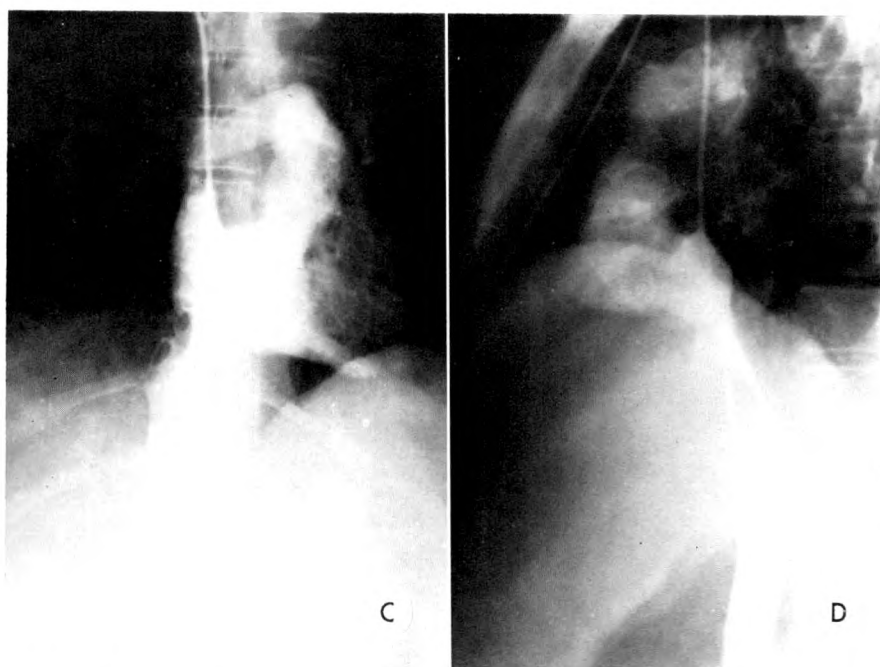
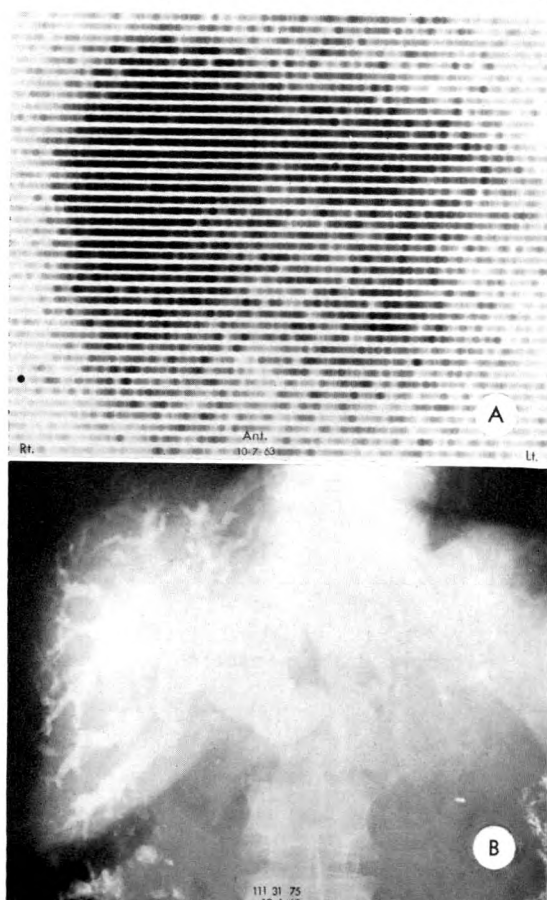
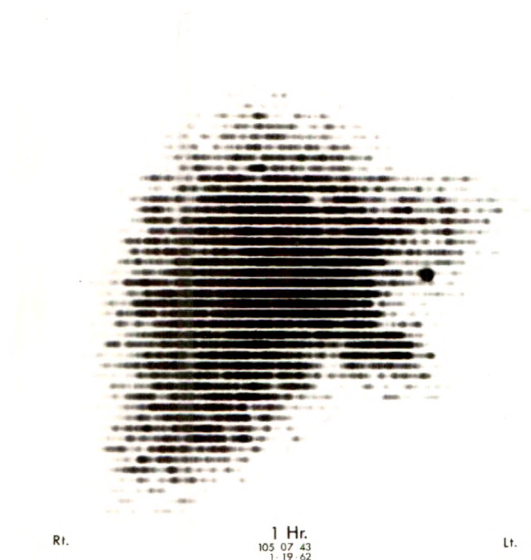


FIG. 3. (C and D) August 13, 1963. Anteroposterior and lateral views of inferior venacavagrams showing the presence of thrombi in the inferior vena cava, hepatic vein area and right atrium.



white female came to the hospital because of gradually increasing intermittent abdominal pain after meals and the appearance of jaundice. The rose bengal hepatic photostan (Fig. 4A) showed a large filling defect in the portal area of the liver with smaller areas of diminished activity radiating from it ("cold" ducts). The liver was moderately enlarged. Figure 4B is a percutaneous cholangiogram demonstrating the configuration of the biliary tree. The correlation with the hepatic photostan is at once apparent.

FIG. 4. Case IV. Carcinoma of the pancreas with extrahepatic biliary obstruction. (A) October 7, 1963. Anterior rose bengal hepatic photostan. Notice the enlarged ducts in the portal area with the radicles of the biliary tree extending from the large main duct. The liver is moderately enlarged. (B) October 1, 1963. Percutaneous cholangiogram. The correlation with the inactive areas in the liver is readily made.



An example of biliary obstruction with "hot" ducts is Case v.

CASE v (Fig. 5). This 63 year old white male presented with recurrent upper abdominal pain.



FIG. 5. Case v. January 19, 1962. Anterior rose bengal hepatic photostan. Obstruction of the common bile duct by stones; 1 hour scan. The intrinsic pattern of the liver is within normal limits for the 1 hour scan, but superimposed on this normal pattern is a dilated duct system which shows hot ducts at this interval. Extending inferiorly and to the left from the lower margin of the liver is the dilated common bile duct.

Serum bilirubin was 6.7 mg. per cent. Physical examination was unremarkable. The obstruction was due to stones in the common bile duct.

If the gallbladder cannot be definitely identified on a liver scan which is started at the top at 20 minutes, it indicates either an abnormality in the function of the liver parenchyma or disease in the gallbladder or duct system. Of 31 patients with extrahepatic obstruction, the gallbladder was

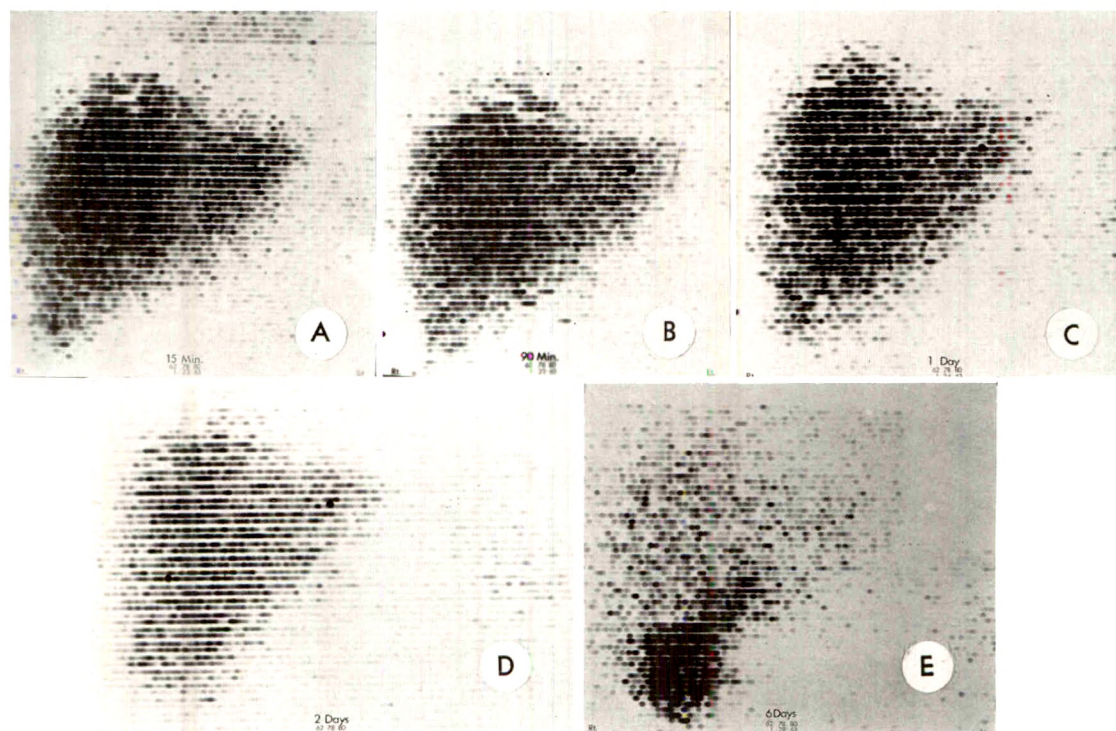


FIG. 6. Case vi. Bile duct carcinoma with extrahepatic obstruction. Serial anterior rose bengal hepatic photostans. (A) January 23, 1963. The liver has a homogeneous pattern. The portal area shows enlarged cold ducts. The heart is seen. (B, C and D) Progress scans at 90 minutes, 1 day and 2 days showing retention of isotope in the liver and the left kidney. (E) Scan at 6 days shows persistence in the liver and left kidney and late activity in the gallbladder.

not shown on the early scans, but in 4 cases it was seen on a delayed scan. In 12 of 25 patients with hepatic parenchymal disease, scans showed the gallbladder. Of 10 patients who had oral cholecystograms that failed to demonstrate the gallbladder, 4 had depiction of the gallbladder by a scan.

The isotope will be found in the small bowel unless there is a complete obstruction to bile flow. The theoretic possibility of a hepatitis so severe that the isotope will not be excreted into the bowel exists but was not encountered in this study. (Two such cases have been seen since the study

was closed.) Occasionally, the only problem in identifying the small bowel is in making the distinction between the small bowel and the presence of radioactive material in the left kidney. Since the right kidney is often obscured by the liver, either serial scans or lateral scans may be required for differentiation.

CASE VI (Fig. 6, A-E). A 69 year old white male entered the hospital because of jaundice. The rose bengal hepatic photoscan showed unusual retention of the isotope in the liver. Scans made at 15 minutes, 90 minutes, 1 day, 2 days, and 6 days are illustrated in Figure 6.

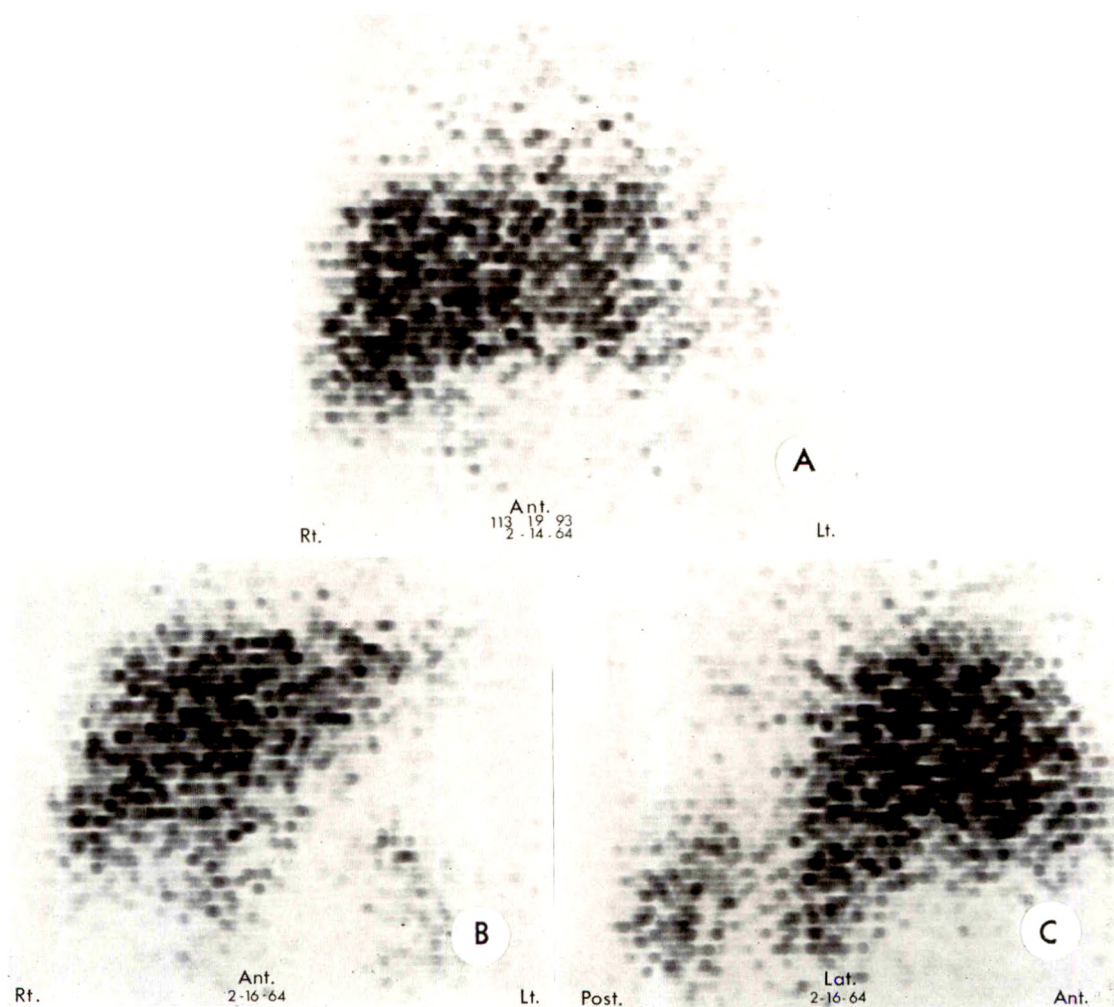


FIG. 7. Biliary atresia and extrahepatic obstruction. (A) February 14, 1964. Anterior rose bengal hepatic photoscan. Some isotope remains in the circulatory system. The liver pattern is homogeneous. (B) Anterior scan at 48 hours. The liver retains the isotope. (C) Lateral scan at 48 hours. There is no isotope in the intestine. The kidneys are shown posteriorly.

TABLE I

SUMMARY OF THE MOST VALUABLE FEATURES OF ROSE BENGAL I¹³¹ HEPATIC PHOTOSCANS

	Diffuse Parenchymal Disease	Localized Parenchymal Disease	Combined Metastases and Extrahepatic Obstruction	Extrahepatic Obstruction
No. of Patients	25	12	7	31
Visualization of Bowel	23 (100%)*	7 (58%)	3 (43%)	19 (61%)
Nonuniform Pattern	17 (68%)	1 (8%)	3 (43%)	6 (19%)
Hepatomegaly	9 (36%)	8 (67%)	2 (29%)	7 (23%)
Visualization of Gallbladder	12 (48%)	4 (33%)	0	4 (13%)
Visualization of Ducts	6 (24%)	2 (13%)	3 (43%)	14 (45%)
Nonuniform Pattern and Visualiza- tion of Bowel	16 (70%)*	1 (8%)	1 (14%)	4 (13%)

* Two scans done early in the series did not cover the area of the small bowel.

The 15 minute scan showed some isotope remaining in the heart. The 90 minute scan did not include enough of the cardiac area to be certain about this feature. All the scans showed some activity in the area of the left kidney and, since no activity was found in the area of the colon on the delayed scans, it was certain that this was the left kidney. The gallbladder filled quite late. This patient was subsequently explored and was found to have adenocarcinoma of the common bile duct which pressed on the cystic duct as well.

The kidneys are ordinarily not identifiable in the absence of moderately severe parenchymal derangement or an extrahepatic obstruction. The kidneys were seen in liver scans in 5 of 25 (20 per cent) pa-

tients with diffuse parenchymal disease, and in 4 of 31 (13 per cent) patients with extrahepatic obstruction.

CASE VII (Fig. 7, A, B and C). A 2 month old female was studied because of neonatal jaundice. The patient has been markedly jaundiced since birth. Serial rose bengal hepatic photoscans were made. The initial scan showed the heart and the kidneys. The scan made 2 days later showed both kidneys well and the lateral scan was made with just enough rotation to show the kidneys posteriorly. The liver was enlarged. The pattern was homogeneous. At operation, no common bile duct or gallbladder was found. A cut in the free edge of the liver did not produce any bile flow and no anastomotic procedure was done.

TABLE II

FINAL DIAGNOSES OF PATIENTS

Diffuse Parenchymal Disease	Localized Parenchymal Disease	Combined Metastases and Extrahepatic Obstruction	Extrahepatic Obstruction
cirrhosis 17	metastases 7	carcinoma of pancreas 3	carcinoma of pancreas 13
hepatitis 7	abscess 3	carcinoma of cecum 1	carcinoma of stomach 1
failure 1	hepatoma 1	carcinoma of lung 1	carcinoma of bile ducts 2
—	infarct 1	metastases 2	carcinoma of gallbladder 1
25	—	—	lymphoma 1
	12	7	biliary atresia 2
			cholelithiasis 10
			choledocholithiasis 1
			—
			31

ANALYSIS OF SCAN FEATURES

A summary of the most valuable features of the scan is presented in Table I. It was found that liver size, intrinsic pattern, demonstration of ducts, gallbladder and small bowel were most helpful in the differential diagnosis while detection of isotope in kidneys, heart and colon was of little significance. The final diagnoses of patients studied are shown in Table II.

SUMMARY

The rose bengal I^{131} hepatic photoscan makes a significant contribution to the differential diagnosis of difficult problems of jaundice. Because the radioactive material can be followed from the circulatory system to the parenchymal structures and into the duct system and the intestinal tract, it provides a unique anatomic and physiologic record. The scan detects local-

ized lesions in the liver such as neoplasm, abscess, infarct or cyst. In most cases it will depict the status of the hepatic parenchyma and disclose the level and degree of any obstruction which may exist.

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A CASE OF MULTI-DIVERTICULAR CYSTIC DILATATION OF THE COMMON AND HEPATIC DUCTS*

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A UNIQUE case of multi-diverticular cystic dilatation of the common and hepatic ducts, a condition which has not been described in the medical literature before, is reported. This condition differs considerably from the classic congenital cystic dilatation of the choledochus. The latter is normally found in females during the first decade of life with symptoms of pain, jaundice and a palpable mass. The multi-diverticular type involves both the common and hepatic bile ducts, presents with abdominal pain as the only clinical symptom and usually appears in an older age group.

The diagnosis in our case was made by oral cholecystography. This is of interest because most cases of congenital cysts are not demonstrated by oral or intravenous cholangiography.

Our patient was treated surgically, which we feel is the treatment of choice and one which can offer significant improvement or cure of this condition. The mortality rate of this operation has steadily decreased from 83 per cent in 1927 to approximately 12 per cent in 1959.

REPORT OF A CASE

Clinical History. In 1961, this 31 year old male complained of recurrent epigastric and left upper quadrant pain of several years' duration. At first, these pains were relatively mild and infrequent. During the last few months, however, they became more frequent and on several occasions were colicky and of short duration. The episodes were not associated with meals, heartburn, jaundice or fever. The bowel movements were normal.

Physical Examination. This revealed mild tenderness over the epigastric and right subcostal regions.

Laboratory Data. The laboratory tests showed a red blood cell count of 5,470,000; hemoglobin 108 per cent or 15 gm., hematocrit 49 per cent, color index 0.93, saturation index 0.91 and a sedimentation rate of 0 in $\frac{1}{2}$ hour and 2 mm. in 1 hour. Leukocytes were 6,800, neutrophils 70 per cent and lymphocytes 30 per cent. The coagulation time was 7 minutes, the bleeding time 65 seconds and the prothrombin time 12 seconds. The prothrombin concentration was 100 per cent, blood urea nitrogen 15 mg. per cent, nonprotein nitrogen 32 mg. per cent, blood sugar 100 mg. per cent, direct bilirubin 0.3 mg. per cent, indirect bilirubin 0.5 mg. per cent and total bilirubin 0.45 mg. per cent. The urinalysis was normal and the stools were positive for ova of ascaris. Except for the presence of ova in the stools, the laboratory tests were considered normal.

Roentgenographic Examination. The routine chest examination and upper gastrointestinal series were negative. Oral cholecystography was performed by administering a double dose of biloptin, approximately 10 hours prior to the examination. The gallbladder was well visualized and of average size and shape. The cystic dilatation of both the common and hepatic bile ducts was faintly demonstrated. Following the administration of sorbitol and cholecystokinin, the gallbladder contracted and the dilated and cystic biliary ducts were much better shown (Fig. 1).

First Operation. On January 28, 1962, Dr. Riveros performed a cholecystectomy. Operative cholangiography at this time demonstrated the multiple cystic changes in the common and hepatic ducts in greater detail (Fig. 2, A and B).

Pathologic Findings. The gallbladder contained normal appearing bile, and showed considerable serosal congestion, thickening of the elastic wall and a velvety mucosa. Histologic sections revealed mucosal papillary projections and edematous thickening of the wall; there was moderate vascular congestion with scat-

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FIG. 1. Marked dilatation and cystic changes in the common and hepatic ducts. Oral cholecystogram made approximately 10 hours after a double dose of biloptin and the administration of sorbitol and cholecystokinin.

tered areas of inflammation, consisting predominantly of lymphocytes and a few polymorphonuclear cells. In addition, there were several areas showing Rokitansky-Aschoff sinuses. The definitive diagnosis was that of acute and chronic cholecystitis.

At operation, the multiple intercommunicating sacs on the hepatic and common bile ducts were easily seen. The common duct drained into the third portion of the duodenum through the sphincter of Oddi. The proximal aspect of the sphincter measured approximately 2 mm. in diameter. The opaque material drained slowly through the sphincter. There was some delay in drainage. The intrahepatic biliary ducts were normal in size.

Second Operation. A second operation was performed on March 7, 1962 which consisted of a valvular choledochojunostomy as well as a complementary jejunojejunostomy. The postoperative course after both the cholecystectomy as well as the choledochojunostomy was uneventful.

Clinical Course. One month postoperatively roentgenograms showed a normally functioning choledochojunal anastomosis. There was evi-

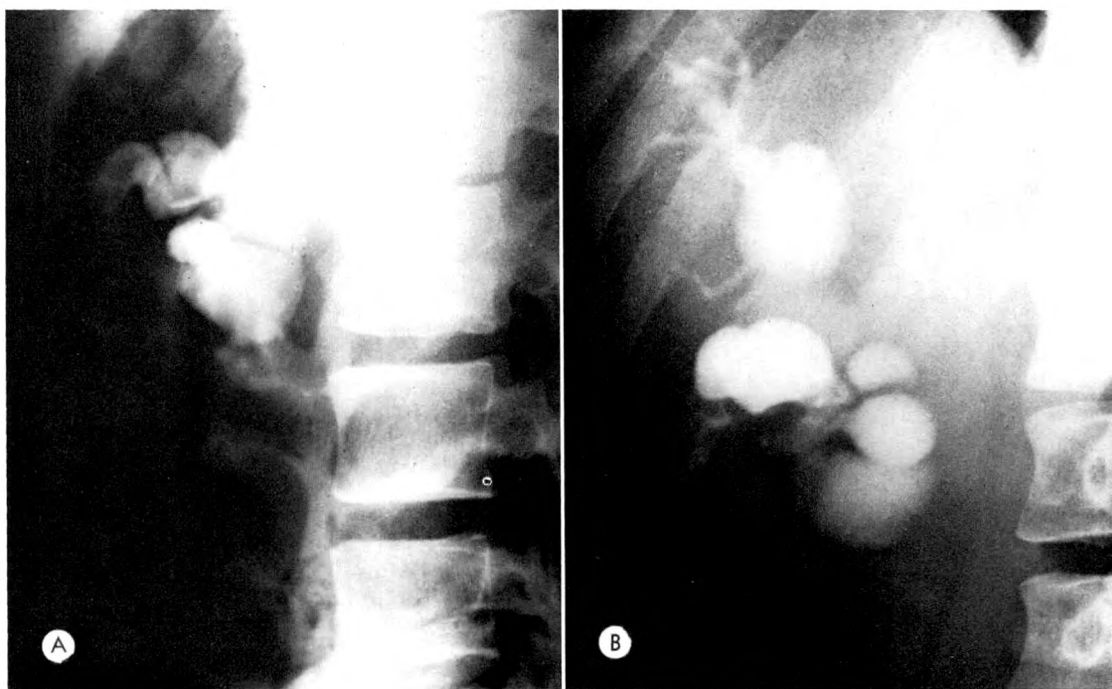


FIG. 2. (A) Lateral operative cholangiogram and (B) oblique operative cholangiogram show the multiple diverticula and the abnormal dilated intrahepatic portion.

dence of air in the common bile duct and of intermittent passage of barium through the anastomotic region. The opening of the choledochus into the third portion of the duodenum was well visualized. A follow-up examination in October, 1962, revealed no significant change in the appearance. The patient has continued to do well to the present time.

DISCUSSION

The roentgenologic and surgical findings in this case were those of a multi-diverticular congenital cyst of the hepatic and common bile ducts. This particular type of anomaly has not been reported in the medical literature.

Of the three types of choledochal cysts as described by Alonso-Lej, Type I is the most frequent and consists of a uniform congenital dilatation of the common bile duct; Type II is actually nothing more than a congenital diverticulum of the common bile duct with a very narrow neck; and Type III, which is a congenital choledochocoele, appears dilated only in the intraduodenal aspect of the duct. We believe that our case may be classified as a variant of Type II, in which the cysts happened to be multiple, not only involving the common but also the hepatic bile ducts.

The only unusual case in the literature which may have some similarity to ours is that reported by Carolli and his collaborators of a 13 year old child who presented with a cholecystic dilatation of the intrahepatic biliary channels. These multiple

ectasias were saccular in appearance and limited only to the subcortical aspect of the intrabiliary radicles. There was uniform dilatation of the hepatic and common bile ducts. Operative cholangiography demonstrated a biliary tree resembling a "fruit tree."

Mallet-Guy believed that the malformation in congenital cystic dilatation is nothing more than a narrowing of the papilla with other changes being secondary to the latter defect. This seems to explain the fact that, even though in our case the common bile duct was vertical and emptied into the most dependent portion of the duodenum, there was, nevertheless, considerable stasis and difficulty in drainage. Carolli and Marcoulides share the same opinion, mainly based on studies from manometric and roentgenographic procedures done during surgery. According to them, the dilatation observed in these cases is only a secondary finding, the primary defect being manifested by narrowing of the lower duct papilla.

SUMMARY

A unique case of multi-diverticular cystic dilatation of the common and hepatic duct is reported.

The oral cholecystographic, operative cholangiographic, surgical and pathologic findings are described.

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THE ROENTGEN FEATURES OF THE MIRIZZI SYNDROME*

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THE Mirizzi syndrome consists of the following elements: (1) an anatomic arrangement of the cystic duct such that it lies almost parallel to the common hepatic duct, (2) impaction of a gallstone in the cystic duct or neck of the gallbladder, (3) partial mechanical obstruction of the common hepatic duct by the stone itself or the resulting inflammatory reaction, and (4) recurrent cholangitis or ultimately cholangitic cirrhosis due to the partial obstruction.¹ We have recognized 2 cases of this syndrome, and they have had strikingly similar roentgen findings on intravenous cholangiography.

REPORT OF CASES

CASE I. A 51 year old woman was hospitalized in August, 1961 for an episode of right upper quadrant pain, chills and fever. Cholecystectomy had been done at another hospital in 1946. At that time a clamp was said to have been placed across the cystic duct and cystic artery at the initial operation, and the gallbladder was removed at a second operation 3 days later. The specimen was reported to show cholelithiasis and chronic cholecystitis. A biliary fistula developed postoperatively. This subsequently closed, but thereafter the patient was troubled by intermittent episodes of right upper quadrant pain, chills and fever which were believed clinically to be due to cholangitis.

Liver function tests were normal except for a bromsulphalein retention of 21.8 per cent and an alkaline phosphatase of 11.3 (normal less than 8.6). Liver biopsy revealed fatty infiltration.

On intravenous cholangiography there was a rounded defect on the lateral aspect of the common hepatic duct with dilatation of the duct proximal to this area (Fig. 1, *A* and *B*). Contrast medium persisted in the proximal common hepatic duct for 4 hours after being cleared from the distal duct.

At operation, it was found that the gallbladder had not been completely removed. A thick-walled, 5 cm. gallbladder remnant with a 1.5 cm. stone impacted in its neck was producing extrinsic compression of the common hepatic duct (Fig. 1*C*).

CASE II. A 66 year old white woman was hospitalized for intermittent fever, fatigue and weakness. Six months earlier she had the sudden onset of painless jaundice and severe pruritus. She was admitted to another hospital at this time where liver function tests showed evidence of obstructive jaundice. The jaundice slowly subsided over a period of 1 week. Two months later severe pruritus recurred but again subsided spontaneously.

Physical examination revealed hepatomegaly and the tip of the spleen was palpable. Laboratory studies included a normal bilirubin, alkaline phosphatase 21.0 Bodansky units, normal SGOT, cephalin flocculation 2+ to 3+, albumin 2.0 gm. per cent, globulin 4.3 gm. per cent. Liver biopsy showed mononuclear infiltrates in the portal zones and slight intracanalicular bile stasis.

A faint, rounded radiolucency was recognized on roentgenograms of the right upper quadrant. An intravenous cholangiogram revealed a rounded defect on the lateral aspect of the common hepatic duct adjacent to this radiolucency and moderate dilatation of the intrahepatic ducts (Fig. 2, *A* and *B*).

At operation there were many adhesions about the gallbladder bed, and the wall of the gallbladder was edematous and hemorrhagic, measuring over 1.0 cm. in thickness. On opening the gallbladder, a large cholesterol stone was found which had eroded the wall of the gallbladder and the adjacent wall of the common hepatic duct (Fig. 2*C*).

DISCUSSION

The broad, curved impressions on the lateral aspect of the common hepatic duct

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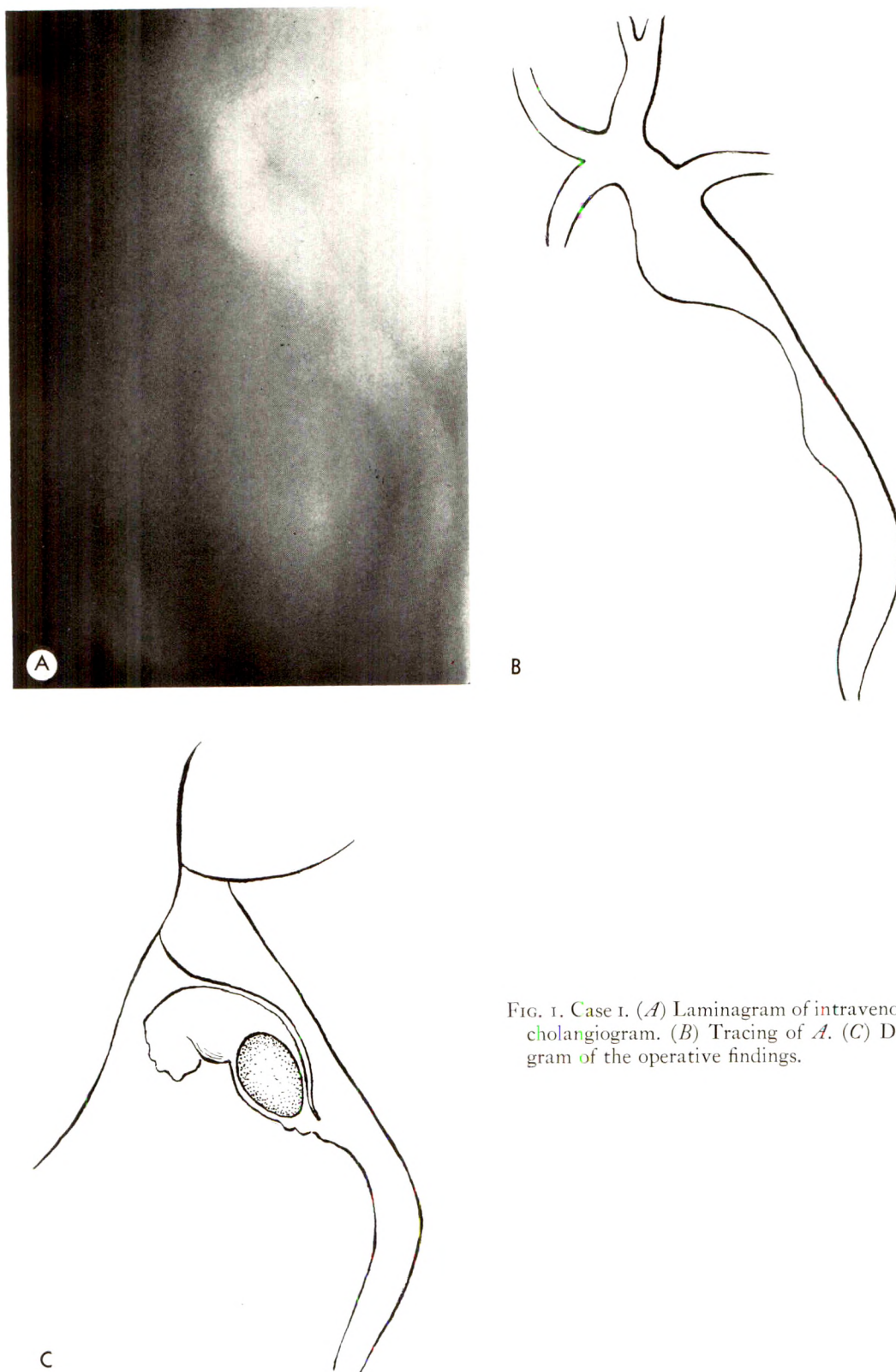


FIG. 1. Case 1. (A) Laminogram of intravenous cholangiogram. (B) Tracing of A. (C) Diagram of the operative findings.

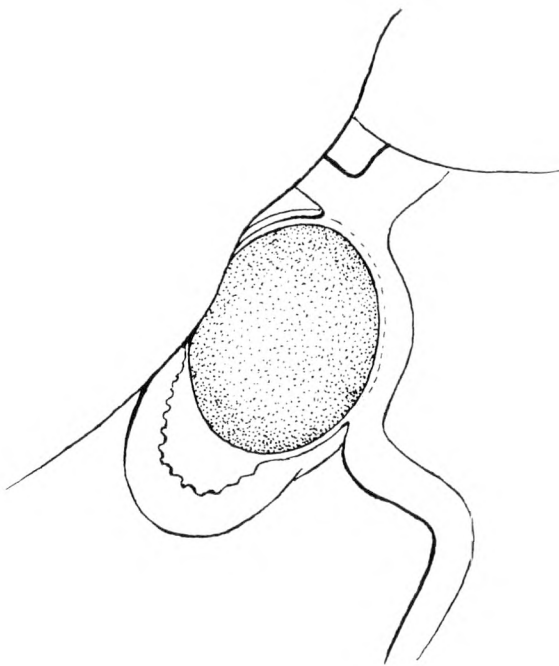
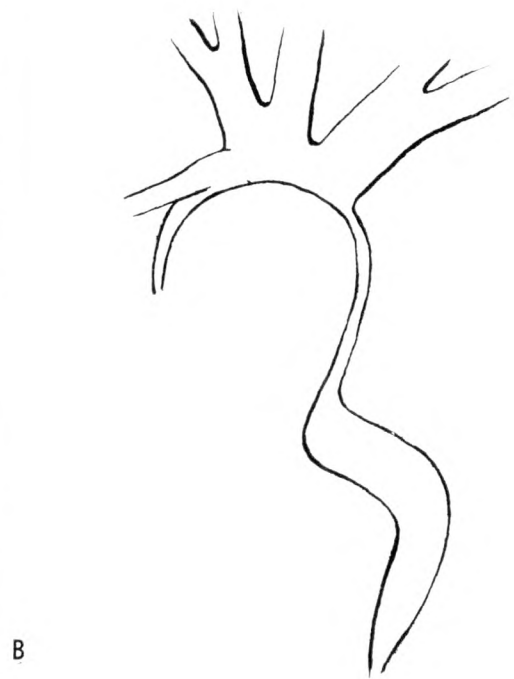
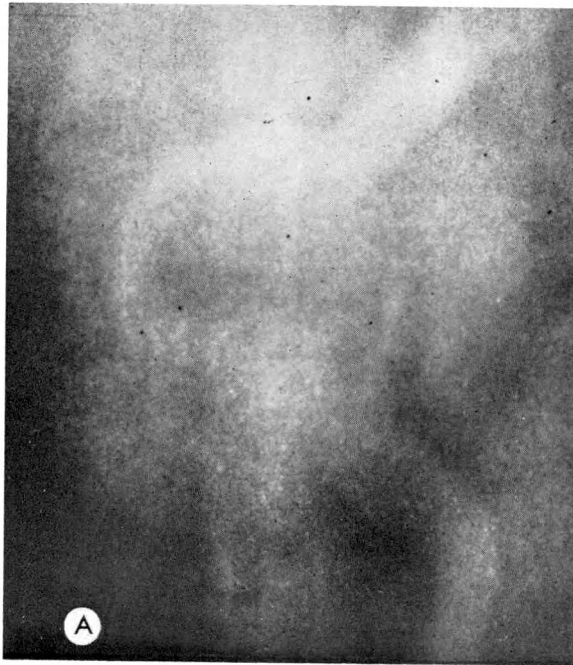


FIG. 2. Case II. (A) Laminogram of intravenous cholangiogram. (B) Tracing of A. (C) Diagram of the operative findings.

in these cases are virtually identical to those described by Dietrich¹ in his report of 4 cases of Mirizzi syndrome. Theoretically, the extrinsic defect need not be from the lateral aspect in view of the possible variations in the relationship of the cystic duct to the common hepatic duct.³ A similar broad, curved impression on the common hepatic duct has been noted to be due to metastatic tumor in the porta hepatis.⁴ In this instance, however, there was concentric narrowing of the duct.

Both patients had roentgenographic and biochemical evidence of partial biliary obstruction. Dilatation of the hepatic ducts proximal to the lesion occurred in both cases, and in Case I delayed clearance of the contrast medium from the common hepatic duct was a striking finding. It has been shown both experimentally³ and clinically⁴ that bile duct obstruction predisposes to bacterial infection of the hepatobiliary system. Clinically, both patients were felt to have recurrent cholangitis, and this impression received support from the liver biopsy findings in Case II.

Finally, while one may decry the perpetuation of eponyms, the term Mirizzi syndrome is useful verbal shorthand to summarize the 4 elements of this entity. It is also fitting that the Argentine surgeon, Dr. P. L. Mirizzi, should be so honored, since he is the acknowledged founder of operative cholangiography.⁶

CONCLUSION

Two cases of Mirizzi syndrome are reported. On intravenous cholangiography both demonstrated broad, curved impressions on the lateral aspect of the common hepatic duct with roentgenographic evidence of partial obstruction of the duct in this area.

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RAPID ORAL CHOLECYSTOGRAPHY*

PHARMACOLOGIC ASSISTANCE TO THE USE OF ORAGRAFIN

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DESPITE the general usefulness and reliability of standard cholecystography techniques (the so-called overnight or 12 to 16 hour technique), a need exists for a rapid oral method for roentgenographic visualization of the gallbladder. A rapid technique has been sought for study of the patient with acute abdominal symptoms, but its greatest use would probably be in adjusting the cholecystographic examination to the convenience of patient and physician, thereby decreasing time and cost of investigation. A successful rapid oral technique depends (1) upon quick arrival of the contrast agent in the duodenum and upper jejunum; (2) a fast rate of absorption; (3) a high differential excretion of the agent into the bile by the liver; and (4) safety and a minimum of side effects for the whole procedure. Iopanoic acid (telepaque) is not suitable for a rapid technique due to slow and incomplete absorption. Recently, other agents which are rapidly absorbed and well excreted in the bile became available. However, even quickly absorbed contrast materials do not afford a successful rapid technique if they are slowly and irregularly moved into the duodenum and jejunum. The time of opacification of bile ducts and gallbladder is dependent upon the emptying rate of the stomach.¹⁷ A solution has been to place the contrast material directly in the small bowel by duodenal intubation, but the method is time consuming for the physician and unpleasant for his patient and not always successful.

The recently introduced cholecystographic agent calcium-bis- β -(3 dimethylamino-methyleneamino-2, 4, 6-triiodophenyl)-propionate, named solu-biloptin in

Europe and oragrafin calcium in the United States, on the basis of pharmacologic studies appears to be well suited to a rapid technique.⁸ Absorption is so rapid that the total output of the compound in bile after intraduodenal administration is of the same order as from intravenous administration.⁸ In rats the intraduodenal administration of 100 mg./kg. of body weight of this compound results in 80 per cent excretion in the bile within 4 hours. The maximum concentration of the compound in the bile was obtained 30 to 90 minutes after administration. Oragrafin calcium is almost a tasteless substance. In animals, its toxicity is of the same order as the other modern cholecystographic agents.⁴

Both the sodium and calcium salts of oragrafin have been reported to be satisfactory cholecystographic agents when utilized in the standard 12 to 16 hour method. In a double blind study, White and Fischer¹⁸ found oragrafin equal to telepaque in the roentgenographic visualization of the gallbladder. Oragrafin, however, was better tolerated by patients and they noted a definite and significantly lower incidence of cramps and diarrhea.

Because orally ingested materials are passed from the stomach into the duodenum irregularly and inconstantly, we were attracted to the use of carbachol-atropine as a pharmacologic aid to gastric peristalsis and emptying.⁵ Dijken and De Wied¹ observed a pronounced increase in gastric peristalsis within 8 to 15 minutes after the subcutaneous injection of 0.4 mg. of carbachol mixed with 0.5 mg. of atropine. Carbachol is a cholinester, exerting a marked cholinergic or parasympathomimet-

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ic effect. It has a strong effect on the musculature of the digestive tract and to a lesser degree on the urinary bladder and heart. Atropine, a parasympatholytic agent, theoretically completely blocks the cholinergic effects of carbachol. In practice, atropine has been known to suppress incompletely the effects of carbachol on the gastrointestinal tract, while controlling other systemic effects of carbachol, such as sweating, heat sensations, salivation and decrease in blood pressure (Fig. 1). Borborygmi and the urge to micturate or defecate are not completely suppressed but are not of sufficient degree to be troublesome. Carbachol-atropine should not be given to patients who are taking other parasympathomimetic drugs such as neostigmine or physostigmine, since the action of the combination could be additive and produce serious cholinergic effects. It should be used with great caution, if at all, in patients with coronary artery disease or severe hypertension or cerebral vascular disease because of possible deleterious muscarinic effects such as fall in diastolic blood pressure, slight tachycardia, peripheral vasodilatation, and so forth. Carbachol is definitely contraindicated in patients with bronchial asthma. The carbachol-atropine should not be given intravenously.

The speed of gastric emptying is known to be directly related to the volume of gastric contents. Accordingly, additional water given along with the cholecystographic agent should be helpful for our purposes.

TECHNIQUE

Patients in the study came from the University Hospital Medical Outpatient Clinic and were not subjected to selection other than exclusion of those in whom the drugs were contraindicated. After their histories had suggested the possibility of gallbladder disease and physical examination had been completed, they were given 6 gm. of oragrafin calcium granules mixed in 200 ml. of water at approximately 10 A.M. All patients had had breakfast. Immediately following the ingestion of the oragrafin and

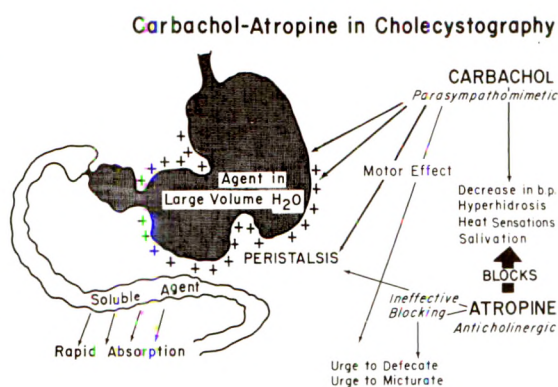


FIG. 1. The effects of carbachol and atropine on the gastrointestinal tract.

water, 0.5 mg. of atropine and 0.4 mg. of carbachol were given subcutaneously in a single injection. Forty-five minutes later, an additional 200 ml. of water was given orally. All other liquids, food and tobacco were not permitted for the first 6 hours of the study. During the first hour and one half after the injection, patients were observed closely by a single observer (W.B.G.) for any possible side effects. Ambulation was allowed, but if the patient desired to recline he was instructed to lie right side down. One and one-half hours after ingestion of the cholecystographic agent, the patient was sent to the Radiology Department.

The 90 minute examination consisted of a 14×17 inch roentgenogram of the entire abdomen plus a roentgenogram of the gallbladder area, utilizing a long cone. If the gallbladder was visualized on the initial films, additional roentgenograms were made at 30 to 60 minute intervals in recumbent, upright or decubitus positions until the gallbladder was considered to have been well visualized and stones diagnosed or excluded. If the gallbladder was not seen initially, additional roentgenograms were made at 1 hour intervals until opacification occurred and then examined further as thought necessary. If no visualization had occurred at 5½ hours, a roentgenogram was obtained at 12 hours if possible; if not, at 24 hours.

TABLE I
FIRST APPEARANCE OF GALLBLADDER OPACIFICATION

0-3 hours	82
3-6 hours	0
6-12 hours	3
12-24 hours	1
Nonvisualization	14
Total cases	100

RESULTS

The time of the first appearance of gallbladder opacification in 100 patients examined by the described technique is shown in Table I. Of the 14 cases in which the gallbladder did not visualize, 3 had no visualization despite observation for 24 hours, 3 despite observation for 12 hours, and 2 for 8 hours. In the remaining 6 patients the diagnosis of nonvisualization is based on shorter observation periods; 3 were examined for 3 hours and 3 for 8 hours.

Table II shows the degree of opacification of the 82 patients in which the gallbladder was seen within the first 3 hours. The classification of 1 to 4+ is more or less standard. We include the class 0 to 1+ for the barely discernible gallbladder, similar to the term "faintly visualizing" gallbladder (Fig. 2, A-E). Increased opacification of the gallbladder after it first visualized was noted in the majority of cases in which such information was sought (Fig. 3). Of the 82 patients showing opacification to some degree within the first 3 hours, 51 were further examined at one or more intervals. Examinations were terminated when the gallbladder was considered sufficiently

TABLE II
DEGREE OF GALLBLADDER OPACIFICATION
WITHIN THE FIRST 3 HOURS

Less than 1 plus	13
1 plus	29
2 plus	30
3 plus	9
4 plus	1
Total cases	82

visualized for diagnosis. In a few instances we could not obtain additional roentgenograms.

The common bile duct was visualized in 37 per cent of patients; visualization occurred in the first 3 hours. There was no correlation between the opaque material remaining in the intestine and nonvisualization of the gallbladder.

SIDE EFFECTS

The side effects following the ingestion of oragrafin calcium and from the subcutaneous dose of the carbachol-atropine mixture were not judged serious in any of the patients. Few patients volunteered complaints. The incidence of effects obtained, mainly by direct questioning, is listed in Table III. No instance of hypotension was found. One patient developed a diffuse pruritic, red maculopapular eruption 8 hours later. This was thought to be a drug eruption from the contrast medium.

DISCUSSION

Our rapid technique must be compared to the few other attempts at rapid cholecystography both as to opacification of the gallbladder and bile ducts and in regard to discomfort and risk to the patient.

TABLE III
SIDE EFFECTS

Type	Per Cent
Urge to micturate	77
Cramping abdominal pain	65
Salivation	48
Urge to defecate	45
Hyperhydrosis	43
Heat sensations	37
Nausea	36
Blurred vision	31
Skin reaction (urticaria) at site of injection	18
"Lightheadedness," "dizzy" or "faint" feelings	17
"Weakness"	8
Vomiting	5
Headache	3
Rhinorrhea	2
Palpitation (no objective finding)	1

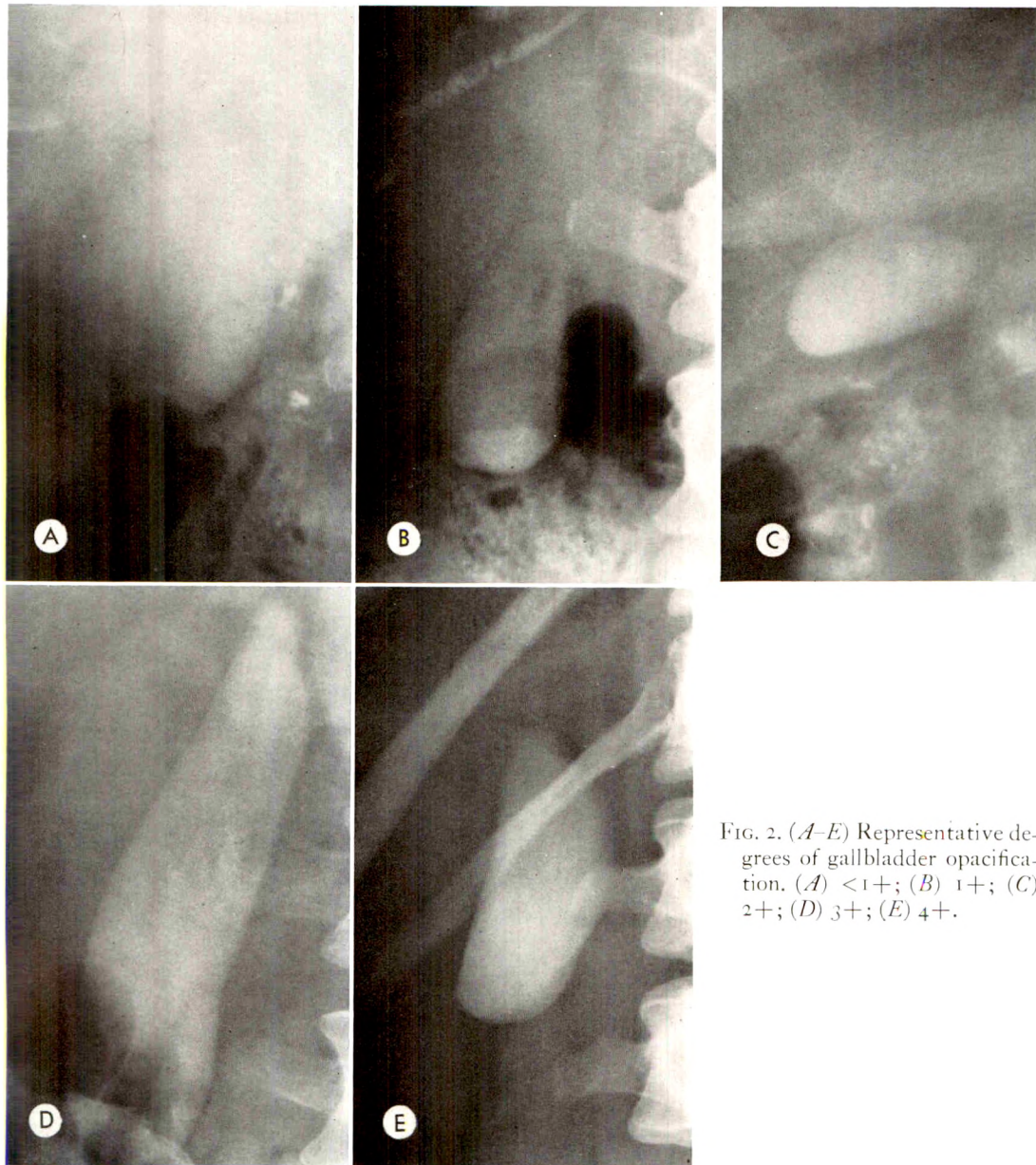


FIG. 2. (A-E) Representative degrees of gallbladder opacification. (A) <1+; (B) 1+; (C) 2+; (D) 3+; (E) 4+.

Madell and Elkin⁹ reported that a 4.5 gm. dose of orablex in fasting patients resulted in only a 29 per cent incidence of satisfactory visualization of the gallbladder at 3 hours. Continuation of the examination to 24 hours raised the incidence of adequate opacification to 70 per cent. The incidence of side effects was not stated.

Four of the newer cholecystographic agents were employed by Nice *et al.*¹¹ in fasting patients, who were placed in the

right lateral decubitus position and given extra water to promote gastric emptying. Good opacification of the gallbladder was obtained in 35.6 per cent of the patients at 2 hours. Visualization was termed fair or poor in 23.5 per cent, while in 40.9 per cent there was no visualization. There were few side effects.

Robinson¹³ reported a group in which 6 gm. of solu-biloptin was given to both fasted and unprepared patients and roent-

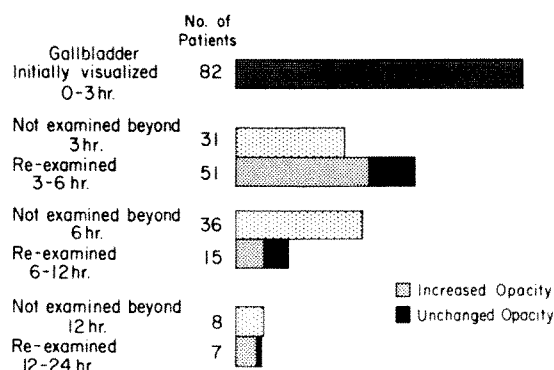


FIG. 3. Graphic representation of the number of patients showing increased opacification with prolonged examination.

genograms were obtained at 3 hours. A glass of cold water was given as an aid to absorption of the contrast agent. Satisfactory gallbladder opacification was seen in 57 per cent of the patients and a faint or no outline in 43 per cent. When 25 patients who had no gallbladder visualization at 3 hours were re-examined by a 12 hour solu-bilopin cholecystogram, 9 were then more adequately seen, 8 of the 9 having normal density and 1 being faintly seen. Of 13 gallbladders faintly visualized at 3 hours, 10 were better opacified by a repeat 12 hour test. Side effects were reported as 2 per cent of the total.

Several other studies are not comparable because they are not truly rapid techniques, but employ in combination a 12 to 16 hour and a 3 hour test.^{2,7,10,16,19,20} In certain other studies a technique similar to that for rapid cholecystography was used for visualization of the common bile duct, primarily in patients who had had cholecystectomy.^{3,14,15}

The present technique for rapid cholecystography with 82 per cent visualization of the gallbladder at 3 hours is certainly an improvement over 3 previous comparable attempts^{9,11,13} but not a fourth.¹² The question to be asked of any rapid technique is how does its accuracy compare with standard cholecystography. The gallbladder visualized satisfactorily for diagnosis in 91 per cent of the cases by a standard cholecys-

tography trial with a single dose of oragrafin at this institution.¹⁸ Another group reports an 84 per cent incidence of gallbladder opacification with oragrafin calcium by a standard technique.⁶ We can say that the incidence of 82 per cent visualization with the present rapid study falls within reasonable limits, and we must look to other criteria to assess the accuracy of our method. Extending the examination for more than 3 hours resulted in visualization of the gallbladder in 4 additional patients. Prolonging the examination to 8 hours in 2 patients, to 12 hours in 3 patients, and to 24 hours in the remaining 3 did not result in visualization of the gallbladder. In 14 cases of nonvisualization, disease of the gallbladder was later substantiated in 6 patients by failure to visualize the gallbladder by single or double dose standard (12 to 16 hours) cholecystography, and in 1 patient by surgery. In only 2 patients a subsequent standard cholecystogram demonstrated the gallbladder. We did not do a subsequent procedure on 5 other patients in whom the gallbladder was not seen by the rapid technique. The assumption that no opacification in 3 hours indicates gallbladder disease need not be made. If the gallbladder opacifies sufficiently for diagnosis at 3 hours, the examination is terminated. If nonvisualization at 3 hours is encountered, the examination can be extended or standard cholecystography can be done at some other time. The latter course is consistent with established medical practice that nonvisualization of the gallbladder on an initial cholecystogram should not be accepted as irrefutable evidence of gallbladder disease, and that failure to visualize must be encountered on the second examination before surgery is contemplated.

Therefore, we feel that the rapid technique employing oragrafin calcium with the assistance of carbachol-atropine is a worthwhile and reliable one, provided its limitations are kept in mind. The majority of patients, those showing adequate opacification of the gallbladder at 3 hours, have the

distinct advantage of a rapid completion of the roentgenologic examination, and thereby a saving of time (and expense) of diagnostic studies can be achieved.

The side effects were not considered serious and we do not think the incidence is excessive in view of the information gained quickly and reliably. We do not accept the present result as ideal, and will continue to search for better means to promote stomach emptying for utilization in rapid cholecystography. The authors recognize the possibility that our results can be related to the patient's ingestion of a moderately large volume of water which is known to promote gastric emptying. The present study is diminished in value by the lack of a control group; that is, a group of patients receiving the same dose of oragrafin calcium but not receiving additional water or carbachol-atropine injection.

Visualization of the common bile duct was not the primary objective of our study. A disappointing 37 per cent for visualization of the duct was obtained, a figure well below previous reports for oral techniques. The technique for rapid visualization of the gallbladder should have resulted in optimum conditions for the demonstration of the common bile duct.

SUMMARY

A new technique for rapid oral cholecystography has been described. This method consists of carbachol-atropine and additional water as aids to gastric emptying so that a double dose of oragrafin calcium may be quickly absorbed from the intestine and excreted in the bile. By this technique, a functioning gallbladder will opacify at 3 hours with a slightly lower incidence than by the standard 12 to 16 hour cholecystography. Nonvisualization can be eliminated in some patients by prolonging the examination beyond 3 hours or by performing standard cholecystography. The side effects encountered, although appreciable in number, were never serious. The examination can be performed in the unprepared patient on the same day of his

initial visit to the physician, thus affording a saving in time, money and inconvenience.

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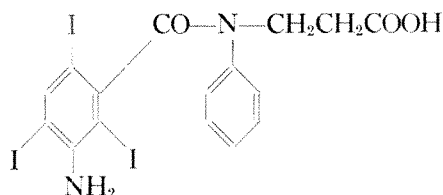
COMPARATIVE EVALUATION OF A NEW ORAL CHOLECYSTOGRAPHIC AGENT U-12,031* WITH TELEPAQUE

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IN EVALUATING a possible new oral cholecystographic agent, we have selected telepaque for a comparison because it has been used widely throughout the United States for several years.

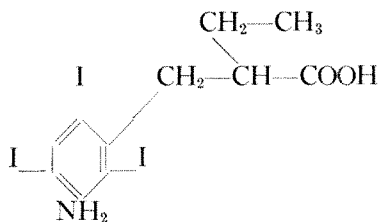
U-12,031 is N-(3-amino-2,4,6-triiodobenzoyl)-N-phenyl-B-amino-propionic acid, and was synthesized as a derivative of 3-amino-2,4,6-triiodobenzoic acid. It is being marketed in Germany and several other European Countries as an oral cholecystographic agent (osbil). Its structural formula is as follows:



Molecular wt. 662

57.5% Iodine

The structural formula of telepaque is as follows:



Molecular wt. 571

66.7% Iodine

The generic name of telepaque is iopanoic acid and the generic name of U-12,031 is iobenzamic acid.

MATERIAL AND METHODS

In this evaluation the "crossover" type of comparison study was done. Each patient received 3 gm. of the contrast agent orally and 1 week later received the other contrast agent. The contrast agent to be given was determined from a table of random numbers. The investigators did not know which compound was being administered. The tests were performed on consecutive outpatients in the Department of Radiology at Charity Hospital, New Orleans. The use of the "crossover" technique eliminates as much as possible patient variation and other variables which are poorly controlled in alternate or random case selection methods.

All patients were carefully questioned regarding side reactions such as cramps, burning, gas formation, urinary symptoms (*i.e.*, burning, frequency, or nocturia), nausea, vomiting, diarrhea, itching, headache, allergy, or burning sensation in chest or throat. The gallbladder visualization was tabulated by two Board certified radiologists and one radiology resident, reading all cases simultaneously. Visualization was tabulated as nonvisualization, faint, fair, good, or excellent visualization. The size of the gallbladder was also evaluated after fatty meal. Visualization of the hepatic,

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cystic and common bile ducts was also tabulated.

Each patient had a urinalysis, serum bilirubin, SGPT, alkaline phosphatase, and blood urea nitrogen studies on the day preceding the first roentgen examination. The above series of tests were also repeated 12 to 16 hours after the administration of each cholecystographic agent, that is, at the time the patient reported for the roentgenographic study. The symptom review for possible side effects was carried out very thoroughly. All subjects with nonvisualization of the gallbladder were interviewed by a psychologist, who used a finger position electrode and a galvanic skin response circuit to determine whether the subjects' responses to questions concerning the ingestion of medication and side effects were truthfully given. Thus, validity of patients' responses was inferred from direct observation of galvanic skin response during the interview. The results indicated that every patient interviewed had actually taken the medications.

The radiopacity of each gallbladder examination was also determined by means of a densitometer so that an objective method of evaluating contrast could be correlated with the more subjective evaluation of the group of radiologists who were interpreting roentgenograms in the usual manner. The height and weight of the subjects were also tabulated. The roentgenographic technique was relatively standard. The kilovoltage varied from 70 to 90 kv. with an average around 80 kv., with 200 ma., 1/2 second, and a 40 inch target-film distance. The film used was Gevaert X-ray Film and for all practical purposes the development conditions were kept constant as all films were developed in a Pako automatic film processor.

In Table I the side effects in 78 patients with iobenzamic acid and 80 patients with iopanoic acid are tabulated. It will be noted that there is relatively little difference between the two. In those patients who received iopanoic acid (telepaque), the incidence of diarrhea was 6 times as great as in

TABLE I
PERCENTAGE INCIDENCE OF SIDE EFFECTS

	Contrast Agent	
	Iobenzamic Acid	Iopanoic Acid
No. of patients	78	80
Average weight	142	151
Gastrointestinal symptoms		
Cramps	16.6%	17.5%
Burning	8.9	11.3
Gas	12.8	15
Nausea	29.4	20
Vomiting	6.4	5
One or more bowel movements	34.6	75
Diarrhea	6.4	38.7
Urinary symptoms		
Frequency	10.5	11.3
Nocturia	17	16.2
Headache	24.4	8.7
Pruritus	10.3	12
Burning in chest or throat	11.5	12

those patients receiving U-12,301 (iobenzamic acid). In those patients receiving iobenzamic acid, the incidence of headache was 3 times that of those patients who received iopanoic acid (telepaque).

Evaluation by the radiologist is given in Table II. There is relatively little difference between the two agents since those studies which are listed as nonvisualization and faint visualization would be considered unsatisfactory from a diagnostic standpoint; whereas those with fair, good, or excellent visualization would probably be satisfactory from a diagnostic standpoint. The percentage of patients showing an excellent visualization was about 10 per cent higher with the use of telepaque. Visualization of the bile ducts was comparable with both agents.

The data revealed by the densitometer studies are listed in Table III. Here again it is to be noted that there is not a very great difference, the median densitometer reading for iobenzamic acid being 1.06 and that of telepaque being 1.14.

Laboratory data are listed in Table IV. It is very significant that slightly over 50 per

TABLE II

SUBJECTIVE ROENTGENOLOGIC EVALUATION

	Contrast Agent	
	Iobenzamic Acid—78 Patients (per cent)	Iopanoic Acid—80 Patients (per cent)
Gallbladder visualization		
Nonvisualization	14.1	13.7
Faint visualization	11.5	11.3
Fair	6.4	8.7
Good	35.9	22.5
Excellent	29.4	40
Bile duct demonstration		
Hepatic	1.3	0
Cystic	16.7	2.5
Common	6.4	5

cent of the patients receiving iobenzamic acid had greater than 25 per cent increase in the serum bilirubin compared with the control value, and over 32 per cent of those receiving iopanoic acid had a similar increase.

When the changes in bilirubin in both the patients receiving osbil and those receiving telepaque were analyzed statistically, it was seen that there was a rise in both groups above the level established for each patient in the pre-study sample of blood. Setting a 95 per cent confidence level (allowing ± 2 standard deviations from the average difference in bilirubin milligrams between the pre-study blood sample and the post-contrast medium sample to give a known accuracy in 95 per cent of the cases), it was seen that a greater number of patients (50 per cent) who had received osbil showed a significant bilirubin rise than those who were administered telepaque (33 per cent).

To arrive at the probability of the rises in bilirubin being due merely to chance or due specifically to the contrast medium administered, the "T-test" for small samples was applied. Taking an average in each group of all the differences in bilirubin from the pre-study samples to the post-study samples and dividing it by the stand-

TABLE III

DENSITOMETRIC READINGS IN CENTER OF GALLBLADDER OPACITY

	Contrast Agent	
	Iobenzamic Acid	Iopanoic Acid
No. of patients	78	80
Mean (average) reading	0.966	0.964
Median (middle) reading	1.06	1.14
Mode (most common) readings	0.4, 0.72 1.75	0.32, 0.79

ard error computed for each group, a probability figure was calculated.

The conclusion was that if osbil was not related to the rises above the pre-study level for that group, the probability of rises as great as these occurring by chance would be one in a million. However, in the other group, the conclusion was that if telepaque was not related to the rises above the pre-study sample, the probability of rises as great as these occurring by chance would be 1 in 2. Therefore, statistically speaking, it appears that osbil was related to the rise in bilirubin level and that telepaque was not.

A statistical analysis of the other laboratory data was also run, but the slight changes in the test results from the pre-study blood samples to the post-contrast medium samples were not significant. In specific gravity, albumin and microscopic analysis of urine samples, no evidence of any abnormality was found that could be

TABLE IV

PERCENTAGE OF PATIENTS SHOWING ABOVE 25 PER CENT INCREASE IN LABORATORY TEST VALUES

	Contrast Agent	
	Iobenzamic Acid	Iopanoic Acid
Serum bilirubin	50.6	32.8
Alkaline phosphatase	5	11.3
SGPT	17.6	17.1
Blood urea nitrogen	8.7	9.7

attributed to ingestion of opaque material.

More intestinal residue was noted following the use of iopanoic acid but in the great majority of cases this in no way interfered with the diagnostic reliability of the examination.

DISCUSSION

From the preceding results, it would seem that there is very little difference in the roentgenologic evaluation and in the presence of side effects when comparing iobenzamic acid with iopanoic acid. Furthermore, the densitometer readings tend to confirm the roentgenologic evaluation. Perhaps of greater significance in this study is the observation that a significant number of patients has an increase in serum bilirubin which is more than 25 per cent above the control value. Ours is one of the largest series in the literature in which a number of blood studies was done on every patient.

Burhenne² reported an increase in serum bilirubin level in 5 of 11 patients following the use of bunamiodyl (orabilex). In the same small group of 11 patients, none showed a rise in serum bilirubin following the use of bilopaque. No other reports^{1,3,4} have given such a significant percentage of changes in the blood chemistry as we have listed in Table IV, and this is especially true in regard to the elevation of serum bilirubin.

The significance of a transient hyperbilirubinemia and its possible relationship to occasional renal failure and hepatic damage is not clear at the present time. Until these phenomena are more clearly understood, it would seem desirable to use a cholecystographic medium without adverse effects on the blood chemistry. It

seems desirable that each contrast agent considered for use should have a fairly complete series of tests done, such as have been done in the present study, to determine to what extent changes in blood chemistry are produced.

SUMMARY

Iobenzamic acid has been compared with iopanoic acid as oral cholecystographic agents in a clinical and roentgenologic study of 80 patients. There is no appreciable difference in the observed side effects, the roentgenographic evaluation, and in densitometry studies.

A statistically significant number of patients had an increase in serum bilirubin following the use of osbil but not telepaque. Other blood chemistry studies also showed a moderate increase in 5-17 per cent of the patients.

More thorough blood chemistry studies are indicated in the evaluation of all oral cholecystographic agents.

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IN VITRO AND IN VIVO VISUALIZATION OF BILIARY CALCULI*

By TOM W. STAPLE, M.D., and WILLIAM H. McALISTER, M.D.
ST. LOUIS, MISSOURI

DEMONSTRATION of biliary calculi on a plain roentgenogram of the abdomen remains the simplest roentgen method for detection of gallbladder disease. Wickbom and Rentzhog⁴ reported the incidence of gallstones that are radiopaque as 19.9 per cent in a series of 722 patients with nonvisualized gallbladders who underwent cholecystectomy. Other authors^{2,3} have reported an incidence of radiopaque gallstones of 20–25 per cent. The purpose of this study was to determine (1) the incidence of radiopaque gallstones both on plain roentgenograms of the abdomen and on *in vitro* studies utilizing water phantoms of different thicknesses, (2) if calcified biliary calculi can be obscured by contrast material, and (3) the percentage of radiopaque stones in each gallbladder examined.

MATERIAL AND METHODS

One hundred and two consecutive resected gallbladders were examined. Each gallbladder was emptied of its stones. These stones were placed at the bottom of beakers filled with 14 cm. and 20 cm. of water. Exposure factors for the 14 cm. phantom were 50 mas. at 55 kv. and for the 20 cm. phantom 50 mas. at 75 kv. The stones were also immersed in a beaker containing a 2 cm. thick layer of 10 per cent methyl diatrizoate.* Films were exposed at 50 mas. and 40 kv. This concentration of methyl diatrizoate was chosen because it best simulated the average density of contrast material seen on oral cholecystograms. The stones of 97 patients were examined in water phantoms and in methyl diatrizoate, while the stones of 5 patients were examined in water phantoms only. In those instances in which

the stones were not visualized when spread out at the bottom of the beaker of water, the calculi were gathered together in a gauze sack and roentgenographed in the 14 cm. phantom to determine any increase in radiopacity under these conditions.

RESULTS

(Tables I and II)

There were a total of 4,611 stones in 102 gallbladders. The stones were either completely opaque, partially calcified, or appeared as radiolucent defects in the water phantoms. In some instances where the stones were not visualized in water, they could be seen when they were gathered together in a gauze sack (Fig. 1, A, B and C).

Of the total number of patients, stones were seen in 49 instances (47 per cent) in the 14 cm. phantom and in 42 instances (41 per cent) in the 20 cm. phantom. However, in 25 of 49 of those with visualization of calculi in the 14 cm. phantom, not all the stones were demonstrated. This occurred in 23 out of 42 in the 20 cm. water phantom. In patients having at least 1 calculus seen in the water phantoms, the per cent of calculi obscured per gallbladder ranged from 13 to 98 per cent in the 14 cm. phantom and from 29 to 98 per cent in the 20 cm. phantom. In 89 (92 per cent) of the patients all of the recovered stones were visualized when placed in a beaker containing methyl diatrizoate. In 7 (7 per cent) a portion of the total number of stones were obscured by the contrast material and in one instance all of the stones were hidden (Fig. 2, A and B; and 3, A and B).

Of the total number of stones, 379 (8 per cent) were seen in the 14 cm. water phantom and 234 (5 per cent) were seen in the 20 cm. water phantom. Of a total of 3,495

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*From The Edward Mallinckrodt Institute of Radiology, Washington University School of Medicine, St. Louis, Missouri. Studies supported in part by a grant from Winthrop Laboratories, New York, New York.

TABLE I
PATIENTS WITH *IN VITRO* VISUALIZATION OF BILIARY CALCULI
Total Patients—102

Phantom	No. of Patients		
	All Stones Visualized	Some of Stones Visualized	Total with Visualization
14 cm. of water	24	25	49
20 cm. of water	19	23	42
2 cm. of contrast material (97 patients)	89	7	96*

* Totally obscured in 1 case.

stones obtained from 97 gallbladders and immersed in methyl diatrizoate 3,246 (93 per cent) were visualized.

Plain roentgenograms were available in 41 patients. Of these, stones were visible in 9 instances (22 per cent). Cholecystograms were available in 54 patients and of these stones were visualized in 42 (78 per cent). The gallbladder was not visualized in 12 of the 54 patients (22 per cent).

DISCUSSION

The signs on plain roentgenograms suggesting gallbladder disease include radio-paque biliary calculi, calcification in the gallbladder wall, milk of calcium bile, and gas in the gallbladder or biliary ductal system. Detection of milk of calcium bile will avoid confusion of this dense substance with contrast material on the cholecystogram. Gas within the gallbladder or biliary duct system suggests the presence of gas-forming bacilli or an abnormal communication between the bowel and biliary system. This may occur either as the result of a cholecystoenterostomy, abnormal patency

of the sphincter of Oddi through surgical intervention, or the passage of gallstones into the bowel.

Although cholecystography has been shown to be from 95 to 99.8 per cent accurate, false negative diagnoses have ranged in varying series from 0.4 to 3 per cent.¹ The principal reasons for false negative examinations are failure to detect gallbladder sand, cholecystitis with cholelithiasis in the presence of a normally visualized gallbladder, faulty cholecystographic techniques, and errors in interpretation of cholecystograms. Wickbom and Rentzhog⁴ cited 25 patients in whom cholecystectomy was performed despite a normal oral cholecystogram. In 4 of these patients stones were found at surgery including 1 patient with 10 stones ranging up to hazel-nut size. They suggested that the contrast material obscured the calculi. *In vitro* obscuration of a portion or all of the gallstones of 8 patients was demonstrated in this study.

Demonstration on plain roentgenograms of the biliary calculi, therefore, is valuable to initially locate calculi, to detect those stones which may be obscured by a contrast material, or to avoid the error of mistaking "calcium bile" for contrast material within the gallbladder.

TABLE II
CALCULI VISUALIZED *IN VITRO*

Total no. of stones	4,611
Visualized in 14 cm. phantom	379 (8%)
Visualized in 20 cm. phantom	234 (5%)
Visualized in 2 cm. 10% methyl diatrizoate	3,246 of 3,495 (93%)

SUMMARY

Biliary calculi from 102 patients were examined roentgenographically in 3 phan-

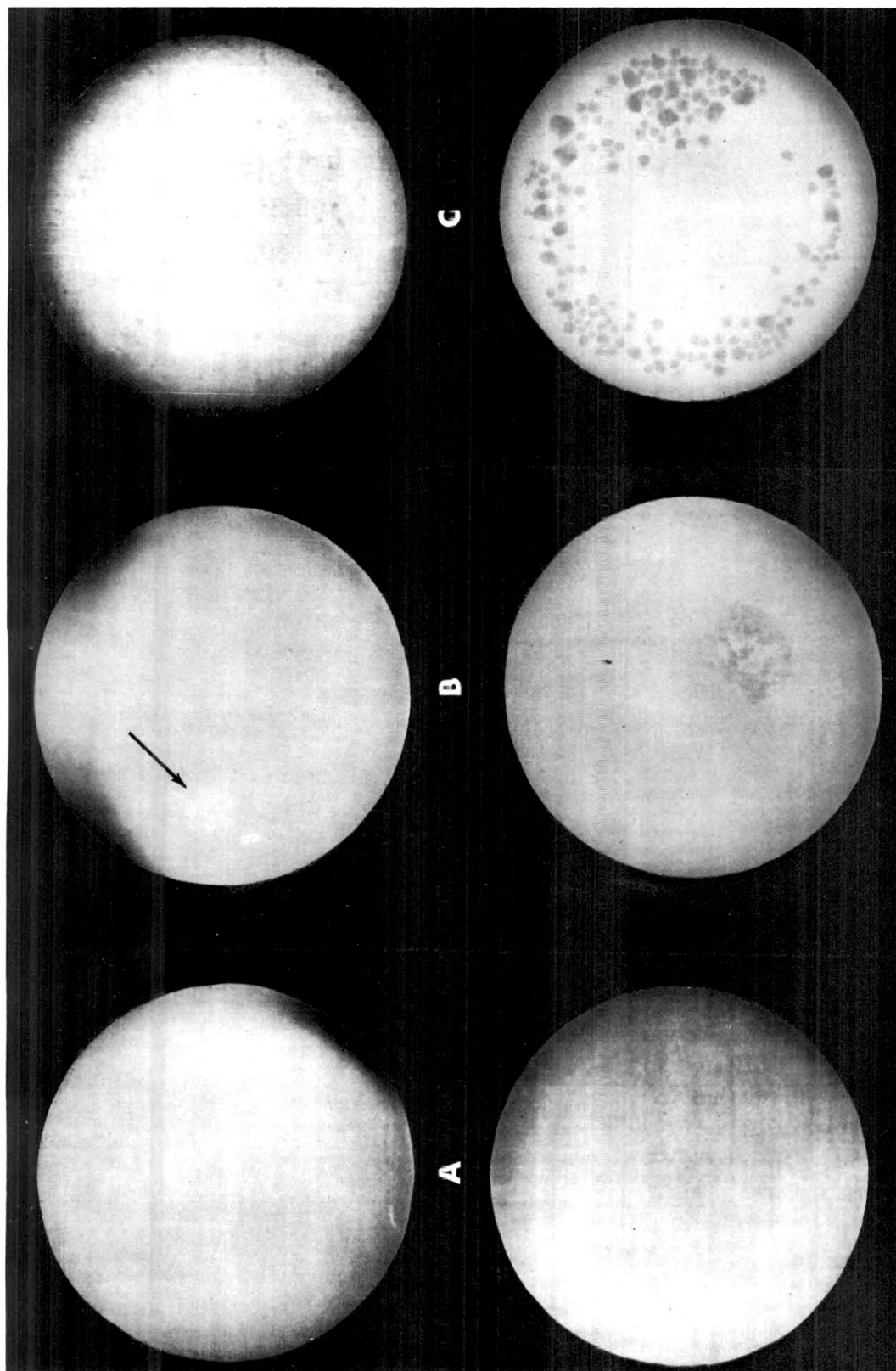


FIG. 1. Visualization of radiolucent calculi by grouping them together in a gauze sack. The calculi are not demonstrated in a 14 cm. water phantom (A). The previously nonvisualized calculi are seen when massed together (B). The same calculi are well visualized in contrast material (C).

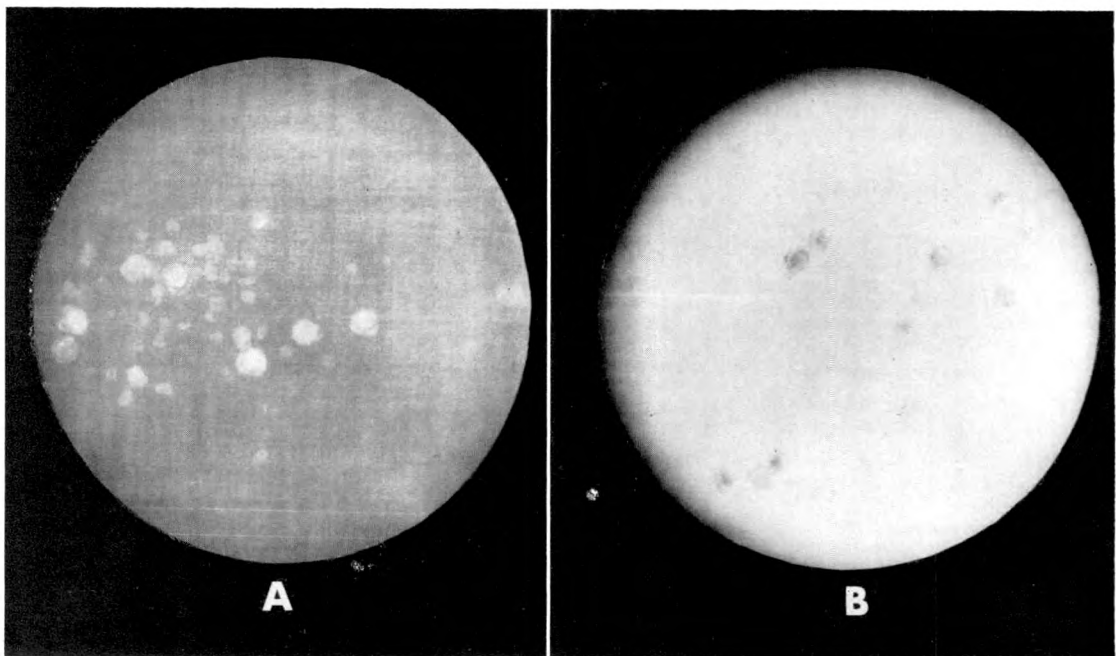


FIG. 2. Partial obscuration of radiopaque calculi by contrast material. Visualization of the total number of calculi in the water phantom (*A*) is markedly reduced by covering these calculi with radiopaque material (*B*).

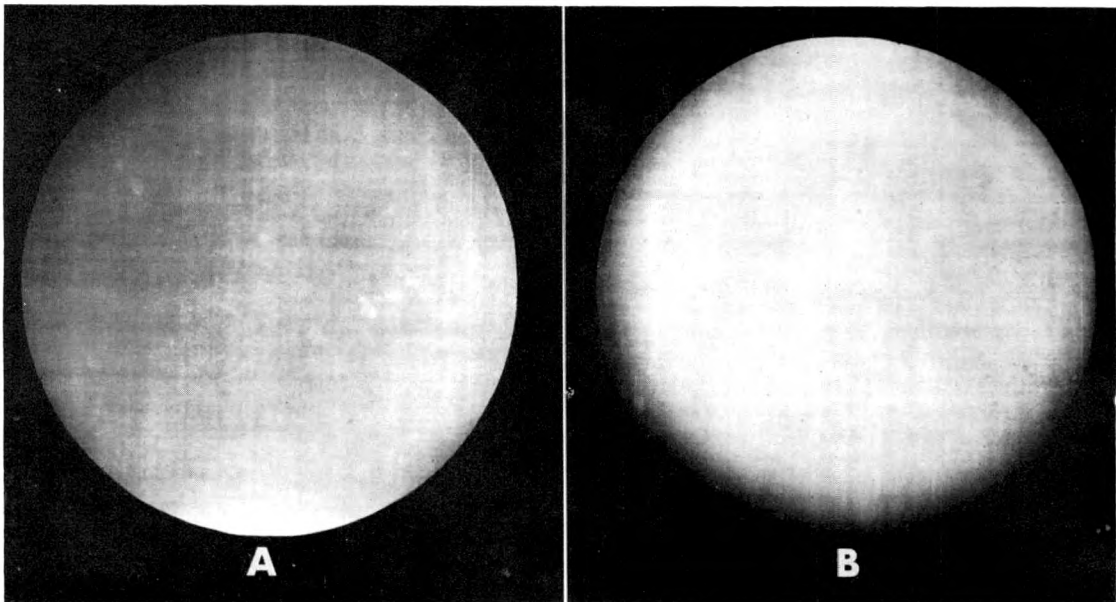


FIG. 3. Complete obscuration of radiopaque calculi. The small calculi seen in the water phantom (*A*) are completely obscured by contrast material (*B*).

toms, 2 containing water of 14 and 20 cm. depth and 1 containing contrast material. Of the patients with available plain roentgenograms of the abdomen or nonfunctioning cholecystograms, 22 per cent had demonstrable radiopaque stones. Forty-seven per cent of the gallbladders contained calculi that could be identified in a 14 cm. water phantom. This percentage fell to 41 when a 20 cm. phantom was used. Eight per cent of the patients had stones that were partially or completely obscured by contrast material *in vitro*. It is therefore recommended, in those patients in whom a highly suggestive clinical history for biliary calculi is elicited and in whom the cholecystogram is normal, that a plain roentgenogram of the gallbladder region after the cholecystopaque has been eliminated from

the gallbladder be obtained to detect any gallstones obscured by the contrast material.

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THE AMERICAN JOURNAL OF ROENTGENOLOGY RADIUM THERAPY AND NUCLEAR MEDICINE

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Forty-eighth Annual Meeting—Golden Anniversary of the American Radium Society: Camelback Inn, Phoenix, Ariz., April 13-16, 1966.

EDITORIAL

THE FORTY-SEVENTH ANNUAL MEETING OF THE AMERICAN RADIUM SOCIETY

THE American Radium Society held its Forty-seventh Annual Meeting in New Orleans, April 8-10, 1965 at the Roosevelt Hotel. The accommodations were superb for both the scientific and social events. The attendance by members and guests surpassed that of the previous year with a total registration of 575, representing 152 members, 256 scientific guests and 167 ladies.

The meeting was officially opened on Thursday morning, April 8 in the spacious Grand Ballroom. Dr. Lewis J. Bristow, Jr., President of the Radiological Society of Louisiana, welcomed the Society to New Orleans and indicated that the local community wished the members to take full advantage of the cultural and recreational facilities. Dr. Joseph H. Farrow of New York delivered the Presidential Address in which he outlined some of the problems facing the medical profession in the diagnosis and treatment of cancer.

He stated that there are now about 1,000 local clinics or tumor services which have been approved by the American College of Surgeons. These cancer activities are distributed throughout this country among more than 5,000 accredited hospitals. It is generally recognized that by cancer-teaching days, by the presentation of problem cases at tumor conferences and by visits of consultants from centers, universities or large metropolitan hospitals, a great deal has been accomplished in the full utilization of local skills and facilities. Furthermore, local interest in diagnosis and treatment has been stimulated and the initial patient-physician relationship maintained.

The increasing number of radical operations which can be performed safely, the

more frequent use of ionizing irradiation and the various methods for palliating advanced cancers have greatly extended the need for accommodations and expensive facilities. Support by grants-in-aid would enable local service to use government funds without federal control. Cancer hospitals and centers were primarily established to render extraordinary services for the cancer patient by the concentration of highly specialized personnel and equipment. They have also functioned as areas for postgraduate training and clinical investigation; in recent years some have expanded into preclinical research centers. Cancer institutes have, for the most part, been concerned with basic research and they provide relatively few, if any, accommodations for patients. In this country, only one of the aforementioned categories is now under complete federal control.

The current recommendations of the President's Commission to conquer heart disease, cancer and stroke are not just a step but, if approved, a tremendous leap by the national government into the diagnostic and therapeutic fields. In order to provide organized leadership and expedite the development of a program to further the control of cancer by available means, there is an urgent need for a committee or association that is truly representative of all the specialties and organizations most active in the diagnosis and treatment of cancer. It should hasten the distribution of up-to-date care for all patients as well as improve the relationship between the medical profession and the public they wish to serve without political pressure.

The first scientific session under the chairmanship of Dr. Robert C. Hickey con-

sisted of presentations on lymphangiography as an adjunct to pelvic radium dosimetry, results of cobalt 60 rotation therapy in carcinoma of cervix without the use of intracavitary radium, carcinoma of the cervix uteri associated with pregnancy and pelvic exenteration for extensive visceral necrosis following radiation therapy for gynecologic cancer.

Following discussion and a short intermission, the second half of the morning's program consisted of presentations on the combined use of irradiation and chemicals in a variety of neoplasms and one contribution on the use of radiotherapy and surgery in carcinoma of the bladder. Dr. Edward T. Krementz of Charity Hospital of New Orleans presented his material on the treatment of retinoblastomas with a combination of intra-arterial injection of T.E.M. followed by irradiation, Dr. Milton Friedman on the use of irradiation and methotrexate in the treatment of squamous cell carcinoma of the head and neck, Dr. Emanuel E. Schwartz on the value of chemotherapy and irradiation of ovarian carcinoma, showing the advantages of ThioTEPA over chlorambucil, Dr. Esmond Millington of Royal Sussex County Hospital in Brighton, England on the combined treatment of carcinoma of the rectum with cobalt 60 teletherapy and 5-Fluorouracil, Dr. David W. Molander on the palliation of metastatic tumors of the soft somatic tissues by the use of irradiation and triple chemotherapy, using methotrexate, actinomycin D, and chlorambucil or cyclophosphamide, and Dr. William Geller on combined therapy in malignant lymphoma using periwinkle alkaloids and methyl hydrazine as adjuncts to proper doses of ionizing radiation.

Dr. Marvin M. D. Williams was chairman of the first half of the second scientific session on Thursday afternoon. The subjects presented included modification of radiation response of tissue by actinomycin D in mice and humans, the radioprotective and antitumor effect of Roche 5-9000 (a new antibiotic), the oxygen factor and tu-

mor volume in C₃H mammary mouse carcinoma system, development of clinical application and technique in hybaroxic radiotherapy, evaluation of lymph node function following irradiation or surgery, the role of regional radiotherapy in organ transplantation by Dr. J. V. Schlosser of the Tulane Transplant Team and comparative study of effects of laser and/or ionizing radiation therapy on experimental malignant tumors by Dr. Paul E. McGuff.

The second half of the afternoon scientific session was devoted to a symposium on radiobiology under the chairmanship of Dr. Juan A. del Regato. The subject of the symposium was variation of cellular radiosensitivity with the mitotic cycle. Dr. L. J. Tolmach spoke on cell generation cycles and methods of synchronizing mammalian cells, Dr. Warren K. Sinclair presented his views on X-ray survival as a function of cell age in its generation cycle, and Dr. Gordon Whitmore presented material on recovery in mammalian cells and possible implications of cell cycle studies in radiotherapy.

In the evening, a reception for members and guests of the Society was held in the attractive Grand Ballroom of the Royal Orleans Hotel, sponsored by the Radium Chemical Company of New York.

The third scientific session on Friday morning was under the chairmanship of Dr. John H. Maus. Dr. Robert C. Hickey was special chairman of a symposium on the management of regional lymph nodes in breast cancer. Dr. George Crile, Jr., of the Cleveland Clinic presented evidence to indicate that simple mastectomy is well adapted to the treatment of certain lesions, modified radical mastectomy with preservation of the muscles can be effectively used in others, and still others are best treated by simple mastectomy plus radiation therapy. He stated that there is mounting evidence that in the early stages of breast cancer uninvolved regional lymph nodes may play a part in the host's immunologic resistance to systemic metastasis and therefore should neither be ir-

irradiated nor removed. Dr. Edgar C. White of the M. D. Anderson Hospital and Tumor Institute presented his views on pre- and postoperative radiation therapy for breast cancer and discussed the four methods which were used from 1948 to 1962 on 1,243 patients. These were: (1) Radical mastectomy was performed when the primary lesion was located in the outer quadrants. If the axillary lymph nodes were free of metastases, no irradiation was added. (2) Radical mastectomy was followed by intensive postoperative irradiation in all cases in which lesions in the outer quadrants were accompanied by axillary lymph node metastases, and in all medially and centrally located lesions regardless of the status of the axillary lymph nodes. (3) Preoperative irradiation followed by radical mastectomy was used when the primary lesion and breast had been grossly disturbed by previous incisions for biopsy specimens, and in the occasional case with borderline criteria for operability. (4) Intensive irradiation was the primary treatment, whether preceded or not by simple mastectomy, in those patients with technically unresectable or clinically inoperable tumors according to the criteria similar to those of Haagensen. Dr. Jerome A. Urban of Memorial Hospital advocated extended monoblock radical mastectomy in an attempt to improve the cure rates of breast cancer. Of 315 patients treated 5 or more years ago, approximately one-half showed axillary involvement and one-third internal mammary involvement. Seventy per cent are alive at 5 years and 66.6 per cent are clinically free of disease. Dr. Ruth J. Guttmann of the Francis Delafield Hospital presented the role of megavoltage irradiation for the control of regional lymph node bearing areas in breast cancer. Regional lymph node bearing areas are routinely irradiated in three situations: (1) in patients who have had a radical mastectomy and where massive involvement of axillary lymph nodes was found; (2) in patients with metastatic or recurrent disease in internal mammary, supraclavicular or axil-

lary lymph nodes after a radical mastectomy; and (3) in patients with a clinically operable lesion who were classified as inoperable because biopsies of the highest axillary or internal mammary lymph nodes showed disease before the mode of treatment was decided upon. The dosage in all patients where definitive radiotherapy is indicated is 5,000 rads delivered in 5 weeks.

Following the session on breast cancer, Dr. Ivor Fix of New York reported on the evaluation of radiotherapy management of intracranial glial tumors without prior surgery.

The second half of the Friday morning session was devoted to subjects on radiation therapy of carcinoma of the vocal cord without mucosal reaction, analysis of complication following megavoltage therapy for squamous cell carcinoma of the tonsillar area, multiple primary cancers of the upper respiratory and alimentary tracts, and some aspects of radiotherapy in relation to thyroid cancer. The latter subject was presented by Professor D. W. Smithers of the Royal Marsden Hospital, London, England.

The fourth scientific session under the chairmanship of Dr. Sidney Rubinfeld on Friday afternoon opened with a panel discussion on the management of solitary metastasis. Dr. Philip Rubin of Strong Memorial Hospital, Rochester, New York, acted as moderator. Other members of the panel were Professor D. W. Smithers, Dr. Danley P. Slaughter of Chicago, Dr. Oscar Creech, Jr., of New Orleans and Dr. Morgan Berthrong of Colorado Springs. This complex subject was discussed by the moderator and panelists in great detail from many standpoints. Many solitary lesions which were considered primary at first proved later to be metastatic after thorough investigation. Professor Smithers emphasized the advantages of delay in the management of lung metastasis.

Following this panel discussion, Dr. Francisco Comas read notes on observation on the accidental prolonged exposure of a family of five to an industrially used co-

balt 60 source. Four persons, including a pregnant young woman, died but the fifth survived and is without symptoms. The family consisted of a father, mother, a boy, a girl and a grandmother. The survivor, the 32 year old father, received a dose of 994-1,716 rems to the whole body. The others who did not survive received higher doses ranging from 1,373 to 5,165 rems. The last paper presented during this session was by Dr. Justin J. Stein on an evaluation of treatment methods for lung carcinoma.

The highlight of the meeting was the presentation of the Annual Janeway Lecture. Dr. Edith H. Ouimby introduced Dr. T. A. Watson, distinguished radiotherapist, who possesses unique experience in the management of cancer. The subject of his lecture was cancer of the breast. In relating his observations in the Province of Saskatchewan, Dr. Watson placed emphasis on the difficulty of staging breast cancer and reporting on results of treatment based on staging. He compared his gross five-year survival rates to those of McWhirter's, believing that the statistics would be more comparable because each received his material from a certain geographic region. His main method of treatment was radical mastectomy followed by postoperative radiotherapy where indicated as compared to McWhirter's main method of treatment which was simple mastectomy followed by postoperative radiotherapy. Dr. Watson's gross five-year survival rates are 58 per cent as compared to McWhirter's 42 per cent. He suggested that further improvement could be obtained by Urban's supra-radical operation combined with postoperative megavoltage irradiation when the lymph nodes are found to be involved.

In the early evening, a reception was held in the spacious and well decorated International Room, which was later followed by the Annual Banquet in the same attractive room. Dr. Joseph H. Farrow introduced the members and wives at the Speaker's Table. Dr. Charles G. Stetson, recent past president of the Society, read and presented a scroll to Mr. Harvey

Picker in honor of his father, Mr. James Picker who, in the past, so generously contributed to the welfare of the American Radium Society. Dr. Farrow, in eloquent terms and with appropriate ceremony, presented the Janeway Medal to Dr. T. A. Watson for his outstanding achievements in the field of radiotherapy. The President also expressed his gratitude on behalf of the Society to Dr. Manuel Garcia and the members of his Local Arrangement Committee for the efforts in extending their gracious hospitality. Members and guests were entertained by talented dancing and singing groups and also participated in the dancing during the evening.

The fifth scientific session was called to order on Saturday morning by Dr. Roald N. Grant of New York serving as chairman. Dr. Edith H. Ouimby moderated a symposium on a critical analysis of some diagnostic tests using radioactive isotopes. Dr. N. David Charkes presented material on bone scanning using Sr^{85} in which he demonstrated metastatic cancer which could not be located by conventional roentgenographic techniques. Other conditions which give rise to new bone formation, such as osteomyelitis, Paget's disease and fracture, also result in marked radiostrontium accumulation, which indicates that a positive scan is not necessarily diagnostic of cancer. Dr. E. Richard King spoke on visualization of the pancreas by means of photoscanning. Seleno⁷⁵-methionine localizes to a certain extent in the pancreas. The interpretation of lesions in the pancreas is still difficult. Preliminary studies have indicated that the pancreas may assume three types; namely, (1) the high transverse, (2) the horseshoe and (3) the sigmoid configuration. Dr. Henry N. Wagner, Jr., demonstrated regional pulmonary blood flow by scanning the lungs after the intravenous injection of I^{131} labeled macroaggregated albumin. Massive pulmonary embolism and other lung lesions causing obstruction to blood flow can be identified with this technique. Dr. Philip M. Johnson contributed information on placental localization by

the use of either human serum albumin labeled with I^{131} or human serum labeled with Cr^{51} . Identification of the placental implantation site facilitates the differential diagnosis of vaginal bleeding during the third trimester of pregnancy and the selection of a satisfactory site for amniocentesis. This procedure proved to have a 97 per cent accuracy as compared to 78 per cent with conventional roentgenography.

The second half of the last scientific session included presentations on cyclotron-produced short-lived radioactive isotopes, the place of lymphangiography in the diagnosis and treatment of malignant disease, hormonal urinary excretion in carcinoma of the cervix, how to irradiate cervix cancer without actually irradiating yourself, evaluation of methods of com-

puter estimation of interstitial and intracavitary dosimetry, cylindroma, and the etiology and pathology of buccal mucosa cancer in South India.

The Forty-seventh Annual Meeting of the American Radium Society proved to be successful by the unusually large attendance, the superb quality of its scientific program and by the social and recreational attractions afforded by the City of New Orleans. The members and guests of the Society owe a debt of gratitude to its President, Dr. Joseph H. Farrow, to Dr. Emanuel Garcia and the members of the Arrangements Committee and to the management of The Roosevelt Hotel for their efforts in providing many excellent conveniences and recreational facilities.

HARRY HAUSER, M.D.



NEWS ITEMS

AMERICAN RADIUM SOCIETY ELECTS NEW OFFICERS

At the Forty-seventh Annual Meeting of the American Radium Society held at the Roosevelt Hotel, New Orleans, La., April 8-10, 1965, the following new officers were elected: *President*: Justin J. Stein, M.D., Los Angeles, Calif.; *President-Elect*: Milton Friedman, M.D., New York, N. Y.; *1st Vice-President*: Manuel Garcia, M.D., New Orleans, La.; *2nd Vice-President*: Richard H. Jesse, M.D., Houston, Texas; *Secretary*: John L. Pool, New York, N. Y.; *Treasurer*: Juan A. del Regato, M.D., Penrose Cancer Hospital, 2215 North Cascade Avenue, Colorado Springs, Colorado 80907.

The Forty-eighth Annual Meeting, celebrating the Golden Anniversary of the foundation of the American Radium Society, will be held at the Camelback Inn, Phoenix, Arizona, April 13-16, 1966.

THE AMERICAN BOARD OF RADIOLOGY

The spring 1966 examination of The American Board of Radiology will be held at the Terrace Hilton Hotel, Cincinnati, Ohio, June 6-10, inclusive. The deadline for filing applications for this examination is December 31, 1965.

The fall 1966 examination will be held at the Washington Hilton Hotel, Washington, D. C., December 5-9, inclusive. The deadline for filing applications is June 30, 1966.

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THE AMERICAN SOCIETY OF NEURORADIOLOGY

The American Society of Neuroradiology

is meeting in Atlantic City, New Jersey on June 11, 1965 at the Dennis Hotel.

For further information, please write to Mannie M. Schechter, M.D., President, Albert Einstein College of Medicine, Eastchester Road, New York 61, New York.

POSTGRADUATE RADIOISOTOPE COURSES IN BRAZIL

The Center of Nuclear Medicine of the University of São Paulo, Brazil, will give a series of Postgraduate Radioisotope Courses in June and extending to September, 1965.

The topics include: Tracer methodology; nuclear medicine; physics applied to radioisotope work and radiation hygiene; radiobiology; and specialty in nuclear medicine.

The courses are sponsored by the Brazilian Ministry of Health.

OAK RIDGE INSTITUTE OF NUCLEAR STUDIES

Fellowships for Graduate Studies in Health Physics

The U. S. Atomic Energy Commission is offering special Fellowships for Graduate Study in Health Physics to 86 selected science students from 27 States and Puerto Rico.

The Commission has also announced the award of 5 new Fellowships in Advanced Health Physics, and the renewal of 6 other Fellowships in the same field of science.

The awards are made under the Special AEC Fellowship Programs in Health Physics and Advanced Health Physics administered for the Commission by the Oak Ridge Institute of Nuclear Studies (ORINS), Oak Ridge, Tenn.

For further information write to: Oak Ridge Institute of Nuclear Studies, P.O. Box 117, Oak Ridge, Tennessee.

BOOK REVIEW

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interest of our readers as space permits.

COBALT-60 TELETHERAPY: A HANDBOOK FOR THE RADIATION THERAPIST AND PHYSICIST. By Ivan H. Smith, M.D., M.Sc., F.R.C.S. (Edin.), F.I.C.S., F.A.C.R., Professor and Head, Department of Therapeutic Radiology, and Lecturer in Pathology, University of Western Ontario; Radiotherapist, Ontario Cancer Foundation London Clinic, London, Canada; (Deceased); J. C. M. Fetterly, M.D., C.M., D.M.R.T.(Eng.), Lecturer in Therapeutic Radiology, University of Western Ontario; Radiotherapist, Ontario Cancer Foundation London Clinic, London, Canada; J. S. Lott, B.A., M.D., D.M.R.T.(Eng.), Associate Professor of Radiology, and Director, Division of Radiotherapy, The Johns Hopkins Medical School and Hospital, Baltimore, Md.; Formerly, Senior Radiotherapist, Ontario Cancer Foundation London Clinic, London, Canada; J. C. F. MacDonald, M.A., Ph.D., Assistant Professor, Therapeutic Radiology, and Honorary Lecturer in Physics, University of Western Ontario; Senior Physicist, Ontario Cancer Foundation London Clinic, London, Canada; Lois M. Myers, B.A., M.D., Ph.D., D.M.R.T.(Eng.), Lecturer in Therapeutic Radiology, University of Western Ontario; Radiotherapist, Ontario Cancer Foundation London Clinic, London, Canada; P. M. Pfalzner, B.A., M.Sc., Lecturer in Therapeutic Radiology, University of Western Ontario; Physicist, Ontario Cancer Foundation London Clinic, London, Canada; and D. H. Thomson, M.B., Ch.B., D.M.R.T.(Eng.), Clinical Associate Professor of Therapeutic Radiology, University of Western Ontario; Senior Radiotherapist, Ontario Cancer Foundation London Clinic, London, Canada. Cloth. Pp. 464, with many illustrations. Price, \$18.50. Hoeber Medical Division, Harper & Row, Publishers, New York, N. Y., 1964.

The authors have conceived a volume which is not intended as a formal treatise on other radiotherapy, but rather an attempt to record the experience of the staff of the Ontario Cancer Foundation London Clinic with the

many phases of cobalt 60 beam therapy. Its contents cover a 10 year period of physical and clinical research on a radiotherapeutic modality which in their belief marks a considerable advance over conventional radiation therapy.

The further object of the volume is to make available the data which the authors have found to be basically essential in cobalt 60 teletherapy. However, the volume is not intended as a text for the medical student or practitioner, but rather as a compendium for the radiation physicist and radiotherapist wishing to assess the value of cobalt 60 beam therapy.

The clinical aspect of the volume is based on more than 4,600 patients treated initially with the original Eldorado cobalt 60 kilocurie stationary unit, and more recently with a rotational hectocurie unit. For each site, treatment policy and case selection are discussed with particular emphasis placed on localization, treatment planning and technique and dosage.

From the standpoint of its usefulness to the radiation physicist, this book is eminently successful. The portions on the physical as well as the technical aspects of cobalt 60 teletherapy are well done and are most helpful to the physicist, particularly the descriptions and references. The basic physics is also well done, but more detailed and comprehensive descriptions can be found in Johns' "The Physics of Radiology." An interesting discussion is presented regarding cobalt 60 production, and the general descriptions of shutters, diaphragms and support mechanisms are well done. There is no doubt, however, that there is a partiality toward the hectocurie unit.

The section on interaction of cobalt 60 gamma rays with matter is very brief.

In the second part of the volume, dealing with the technical aspects of cobalt 60 teletherapy, the physical data required for treatment are summarized succinctly. The suggestions, with regards to tumor localization and methods of beam direction, are most helpful in terms of treatment planning. There is an excellent discussion of dose homogeneity, with a good basic summary regarding treatment planning itself. The sections dealing with fixed

beam therapy and moving field therapy are good but too brief and the same criticism can be made of the section on wedge filters.

Evaluation of patient results, presented in the clinical aspects, is primarily the impression of the authors, and the comparisons do not come from controlled clinical trials of cobalt 60 teletherapy versus conventional radiation therapy. The treatment policy adopted for the cobalt 60 techniques is essentially the same as that already successfully established for conventional roentgen therapy. It is the belief of the authors that this modality of treatment has replaced interstitial radium application in certain sites, particularly the buccal mucosa, tonsil and maxillary antrum. They also conclude that preoperative radiation therapy can be better carried out using the cobalt 60 devices. Their final conclusion is that cures may not be appreciably improved, but certainly there are major advances that accrue to the radiotherapist utilizing this modality.

In the section dealing with the additional physical data, there are many good and useful suggestions regarding cobalt 60 installations, the problems of penumbra, build-up to electronic equilibrium, etc. The appendices also are well done and most helpful.

In summary, the authors have produced a work that is superior in terms of physics, giving general excellent descriptions and a large compendium of references which will be most useful to the clinical radiotherapist.

LUTHER W. BRADY, M.D.

ROENTGENOGRAPHY AND ROENTGENOLOGY OF THE MIDDLE EAR AND MASTOID PROCESS. By Lewis E. Etter, B.S., M.D., F.A.C.R., Professor of Radiology, Western Psychiatric Institute and Clinic, The Falk Clinic School of Medicine, University of Pittsburgh; Consultant, C. Howard Marcy State Hospital, Pittsburgh, Pa. With the collaboration of Lawrence C. Cross, R.T., Chief Research X-Ray Technician, Western Psychiatric Institute and Clinic; Chief X-Ray Technician, Veterans Administration Hospital, Leech Farm, Pittsburgh, Pa.; and Merle J. Stuart, R.T., Research X-Ray Technician, Western Psychiatric Institute and Clinic, The Falk Clinic School of Medicine, University of Pittsburgh, Pittsburgh, Pa. With a foreword by Kenneth M. Day, M.D. Cloth. Pp. 149, with 134 figures. Price, \$9.75. Charles C

Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Ill., 1964.

A satisfactory examination of the mastoid process and of the middle ear should include, according to Dr. Etter, six classic projections: The Schüller's, the Owen's, the Stenvers', the Mayer's, the Chamberlain-Towne's and the basal or submentovertical projection.

In his monograph, Dr. Etter reviews for each projection the technique and the normal roentgenographic anatomy, as demonstrated in the dry skull and in living subjects. A few pathologic examples are shown for each projection.

The last part of the book deals with the principles, the technique and examples of direct roentgenographic enlargement with four-fold magnification, which Dr. Etter uses for the study of the mastoid process and of the ear.

The volume, which should be considered an atlas in view of the large number of excellent illustrations, is dedicated to all radiologists and X-ray technicians desirous of obtaining the maximum amount of information available from standard roentgenography and conventional equipment.

GALDINO E. VALVASSORI, M.D.

BOOKS RECEIVED

TWO CENTURIES OF MEDICINE: A HISTORY OF THE SCHOOL OF MEDICINE, UNIVERSITY OF PENNSYLVANIA. By George W. Corner, M.D., Sc.D., LL.D. Cloth. Pp. 363, with some illustrations. J. B. Lippincott Company, Philadelphia 5, Pa., 1965.

TUMOURS OF THE KIDNEY AND URETER. Edited by Sir Eric Riches, M.C., M.S., F.R.C.S., Emeritus Urological Surgeon, The Middlesex Hospital; Urologist, St. Andrew's Hospital, Dollis Hill; Past Vice-President, The Royal College of Surgeons of England. Volume V, of Monographs on Neoplastic Disease at Various Sites. General Editor, D. W. Smithers, M.D., F.R.C.P., F.R.C.S., F.F.R. Cloth. Pp. 416, with 200 illustrations. Price, \$14.50. The Williams & Wilkins Company, Baltimore 2, Md., 1964.

RADIATION ACCIDENTS AND EMERGENCIES IN MEDICINE, RESEARCH, AND INDUSTRY. Edited by Lawrence H. Lanzl, Ph.D., Department of Radiology and Argonne Cancer Research Hospital, The University of Chicago, Chicago, Ill.; John H. Pingel, M.P.H., Industrial Hygiene and Safety Division, Argonne National Laboratory, Argonne, Ill.; and John H. Rust, Ph.D., Section of Nuclear Medicine, Department of Pharmacology, The University of Chicago, Chicago, Ill. Cloth. Pp. 328,

- with some figures. Price, \$11.75. Charles C Thomas, Publisher, 301-327 East Lawrence Avenue, Springfield, Ill., 1965.
- THE RADIOLOGIC CLINICS OF NORTH AMERICA.** Symposium on The Urinary Tract. Guest Editor, Isadore Meschan, M.D. April, 1965, Volume III, Number 1. Cloth. Pp. 177, with many illustrations. W. B. Saunders Company, West Washington Square, Philadelphia, Pa., 1965.
- ROENTGEN DIAGNOSIS OF THE HEART AND GREAT VESSELS.** Second American edition, enlarged revision. By Erich Zdansky, M.D., Professor of Roentgenology, University Institute for Roentgen Diagnosis and X-ray Therapy, Basel, Switzerland. Translated by Linn J. Boyd, M.D., F.A.C.P., Clinical Professor of Medicine; Consultant, School of Graduate Sciences, New York Medical College, Flower and Fifth Avenue Hospitals, New York, N. Y. Cloth. Pp. 423, with 386 figures. Price, \$26.50. Grune & Stratton, Inc., 381 Park Avenue South, New York 16, N. Y., 1965.
- VASCULAR ROENTGENOLOGY: ARTERIOGRAPHY, PHLEBOGRAPHY, LYMPHOGRAPHY.** Edited by Robert A. Schobinger, M.D., F.A.C.A., F.I.C.A., S.G.P., Diplomate, American Board of Surgery; Diplomate F.M.H. in Surgery, Switzerland; Department of Surgery, Clinic St. Anna, Lucerne, Switzerland; and Francis F. Ruzicka, Jr., M.D., F.A.C.R., Director, Department of Radiology, The St. Vincent's Hospital and Medical Center, New York; Clinical Professor of Radiology, New York University School of Medicine, New York. Cloth. Pp. 747, with many illustrations. The Macmillan Company, 60 Fifth Avenue, New York, N. Y., 1964.
- CONGENITAL HEART DISEASE: CORRELATION OF PATHOLOGIC ANATOMY AND ANGIOCARDIOGRAPHY.** By Jesse E. Edwards, M.D., Director of Laboratories, The Charles T. Miller Hospital, St. Paul, Minn.; Clinical Professor of Pathology, Medical School and Professor of Pathology, Graduate School, University of Minnesota, Minneapolis, Minn.; Lewis S. Carey, M.D., Director, Cardiovascular Diagnostic Unit, Department of Radiology, St. Joseph's Hospital, St. Paul, Minn.; Clinical Assistant Professor, Department of Radiology, University of Minnesota Medical School, Minneapolis, Minn.; Henry N. Neufeld, M.D., Chief, Heart Institute, Tel-Hashomer Government Hospital, Israel; Formerly Research Associate, The Charles T. Miller Hospital, St. Paul, Minn.; Honorary Fellow, University of Minnesota, Minneapolis, Minn.; and Richard G. Lester, M.D., Chairman, Department of Radiology, Medical College of Virginia, Richmond, Va. In Two Volumes. Cloth. Volume 1, pp. 1 to 396; Volume 11, pp. 397 to 890, with many illustrations. Price, \$45.00 the set. W. B. Saunders Company, West Washington Square, Philadelphia, Pa., 1965.
- PROGRESS IN CLINICAL CANCER.** Volume I. Edited by Irving M. Ariel, M.D., F.A.C.S., Associate Clinical Professor of Surgery and Attending Surgeon, New York Medical College, Flower and Fifth Avenue Hospitals; Attending Surgeon and Chief of the Soft Somatic Tissue Tumor Service, Hospital for Joint Disease; Attending Surgeon, Pack Medical Group, New York, N.Y. Cloth. Pp. 780, with many illustrations. Price, \$35.00. Grune & Stratton, Inc., 381 Park Avenue South, New York 16, N. Y., 1965.
- SELECTED PAPERS FROM THE INSTITUTE OF CANCER RESEARCH: ROYAL CANCER HOSPITAL AND FROM THE ROYAL MARSDEN HOSPITAL, 1963.** Published by order of the Committee of Management of the Institute and the Board of Governors of the Royal Marsden Hospital, London. Volume 18. Cloth. Pp. 1,425, with many illustrations. The Chester Beatty Research Institute, Fulham Road, London, S.W.3, England, 1965.
- EARLY DIAGNOSIS, PATHOHISTOLOGY AND TREATMENT OF MALIGNANT TUMOURS OF THE SKIN.** By T. Venkei, M.D., and J. Sugár, M.D., State Institute of Oncology, and Research Institute of Onco-pathology, Budapest. Cloth. Pp. 368, with 324 figures. Price, \$10.00. Akadémiai Kiadó, Publishing House of the Hungarian Academy of Sciences, Budapest V. Alkotmány U. 21., 1965.



ABSTRACTS OF RADIOLOGICAL LITERATURE

INDEX TO ABSTRACTS

ROENTGEN DIAGNOSIS

HEAD

- VARADARAJAN, M. G., and RAMAMURTHI, B.: Assessment of normal physiological intracranial calcification occurring in Indian subjects..... 511
- MADONIA, G., and MARLEY, A.: The auditory ossicles of several mammals studied by combined radiographic enlargement..... 511
- LEICHSENRING, F.: Pathological changes in the cervical vertebrae in patients who died after head trauma..... 511
- vara (osteochondrosis deformans tibiae): a survey of seventy-one cases..... 516
- GOLDENBERG, R. R.: Chondrosarcoma..... 516
- NORMAN, W. H.: A child with three lower extremities..... 517
- HORWITZ, T.: Some pathologic considerations in the diagnosis of lumbar intervertebral disk lesions..... 517

GENERAL

- BROWN, J.: Primary amyloidosis..... 517

RADIATION THERAPY

- GRAYSTON, J. T. *et al.*: Mycoplasma pneumoniae infections: clinical and epidemiologic studies..... 511
- METYSŠ, R. *et al.*: Pulmonary chondromatous hamartomas..... 512
- HOLMES, B. R.: Calcifications of the heart..... 512
- VASTERLING, H.-W., and HILFRICH, H.-J.: Prognostic significance of temperature elevation during radiation therapy of carcinoma of the uterine cervix..... 518
- BONO, F., and DETTORI, P.: Angiographic and encephalographic findings in cerebral tumors following high energy radiation therapy.... 518
- LAMP, J. C. *et al.*: Keratoacanthoma of the subungual region: a clinicopathological and therapeutic study..... 518

ABDOMEN

- GLANVILLE, J. N.: Further experience with the ward acute barium meal..... 513
- INDRA, K. J., and CHAWLA, S.: Trichobezoar—a case report..... 513
- MORGENSTERN, L., FREILICH, M., and PANISH, J. F.: The circumferential small-bowel ulcer: clinical aspects in 17 patients..... 513
- SOUTER, W. A.: Bolus obstruction of gut after use of hydrophilic colloid laxatives..... 514
- DASSEL, P. M.: Intestinal obstruction—a review of some uncommon causes..... 514
- MARCUS R., and WATT, J.: The radiological appearances of diverticula in the anti-mesenteric intertenia area of the pelvic colon..... 514
- McAFEE, J. G. *et al.*: Tc^{99m} pertechnetate for brain scanning..... 519
- SCHMIDTKE, I., BLATTNER, L., and SCHIESSLE, W.: Autoradiographic observations after inhalation of radioactive yttrium and zirconium..... 519

CHEMOTHERAPY

- HUBACHER, O.: Treatment of the terminal stages of cancer with SP-G and SP-I: preliminary communication..... 520

MISCELLANEOUS

- CLAUS, H. G.: Does a striated type of osteopokilosis exist?..... 515
- CHAWLA, S.: Cranio-skeletal dysplasia with acro-osteolysis..... 515
- LANGENSKIÖLD, A., and RISKA, E. B.: Tibia
- LINDBLOM, A. F.: Measuring of doses received by the personnel in radiological departments and its importance..... 520
- WACHSMANN, F., and KERMANI, D.: Experiments on the biologic effect of extremely high radiation doses..... 520

ROENTGEN DIAGNOSIS

HEAD

VARADARAJAN, M. G., and RAMAMURTHI, B.

Assessment of normal physiological intracranial calcification occurring in Indian subjects.

Mediscope, Madras, Dec., 1964, 7, 453-466.

(From: The Barnard Institute of Radiology, and the Govt. Gen. Hospital, Madras, India.)

Physiologic intracranial calcifications include deposits within the pineal gland, the choroid plexus, the dura mater, the pacchionian bodies, and the arteries. These calcifications in and of themselves may be of little importance, but they can be of great aid in diagnosing other intracranial diseases, and they must be differentiated from pathologic conditions.

Pineal calcification represents a normal degenerative change in a structure which in fetal life and infancy is histologically active but whose function is as yet unknown. A pineal tumor may be suspected if the diameter of the calcified area is greater than 1 cm. In most of the reported series the incidence of pineal calcification has been between 50 and 65 per cent for all ages, there being a gradual increase from puberty through old age. However, in South Indian subjects the incidence has been no higher than 8 per cent. The authors suggest that lower nutritional standards may be a factor.

Choroid plexus calcifications occur 2.5 cm. on either side of the midline, and they have been reported in the range of 8 to 25 per cent of all skulls. In this South Indian series the incidence of such calcification was again low, varying between 0.5 and 3.0 per cent.

Dura mater calcification in the falx can continue to true bone formation. It is frequently asymmetric, but because of its rigidity it is of little use in detecting space occupying lesions. It occurs in about 9 per cent of all skulls, but in this South Indian series it was found in only 3.8 to 5.8 per cent.

Interclinoid bridging and tentorial calcification were other less frequent calcifications observed.—*Arch H. Hall, M.D.*

MADONIA, G., and MARLEY, A. Gli ossicini dell'udito di alcuni mammiferi studiati con ingrandimento radiografico combinato. (The auditory ossicles of several mammals studied by the combined radiographic enlargement.) *Radiol. med.*, Sept., 1964, 50, 886-899. (Address: Dott. Gino Madonia, Via Folla di Sotto 20/E, Pavia, Italy.)

The auditory ossicles of several mammals have been studied by the authors by a combined radiographic and photographic enlargement technique, allowing a tenfold magnification.

The morphology of the malleus, incus and some-

times of the stapes of the ox, lamb, chamois, roebuck, pig, horse, mule, dog, fox, cat, rabbit, guinea pig and beaver is accurately described.

The investigation proves that the auditory ossicles of the mammals (including the man) are quite similar, although particular features typical of each animal have been found. These variations involve especially the processes for the suspension of the ossicular chain to the tympanic cavity. As a result, these changes may affect the oscillation of the ossicular chain to sound waves.—*Galdino E. Valvassori, M.D.*

LEICHSENKING, F. Pathological changes in the cervical vertebrae in patients who died after head trauma. *German Med. Monthly*, Jan., 1965, 50, 17-25. (From: Institute of Pathology, Barmbek General Hospital, Hamburg, Germany.)

The author investigated the cervical spine of all those who died with unequivocal skull injuries during the last few years. The study comprises 20 such cases coming to necropsy; they were divided into 4 categories according to the severity of the head trauma.

The first group included persons with occipital fractures; the second those with non-occipital fractures of the base of the skull; the third, persons with fracture lines of the skull excluding its base; and the fourth group those with soft tissue injuries of the head but no fractures.

The necropsy findings showed clearly that abnormalities of the cervical spine were consistently present in all cases of head injuries regardless of whether these were associated with fractures. In each case more or less extensive hemorrhages were demonstrable in the connective tissues and paravertebral musculature as well as between the spinous processes. Traumatic hemorrhages were seen in the intervertebral disks.

There were peridural hemorrhages extending into the intervertebral foramina in the majority of cases and disseminated petechiae with associated edema of the central canal and spinal leptomeninges were demonstrated as well. These hemorrhages can occur in the absence of severe cerebral trauma. Therefore, in each case of head trauma the possibility of involvement of the cervical vertebrae and surrounding structures must be kept in mind.—*Francis Shea, M.D.*

NECK AND CHEST

GRAYSTON, J. THOMAS, ALEXANDER, E. RUSSELL, KENNY, GEORGE E., CLARKE, EDMUND R., FREMONT, JOSEPH C., and MACCOLL, WILLIAM A. Mycoplasma pneumoniae infections: clinical and epidemiologic studies. *J.A.M.A.*, Feb. 1, 1965, 191, 369-374. (Address: Dr. Grayston, Department of Preven-

tive Medicine, University of Washington School of Medicine, Seattle, Wash. 98105.)

Atypical pneumonia was a term applied to pneumonia which is caused by a number of etiologic agents, including multiple virus diseases. Out of this group such distinct entities as influenza, psittacosis, Q-fever, and adenovirus pneumonias could be identified. The etiology of the large remaining group, however, was in doubt until some workers were able to confirm the Eaton agent etiology. In 1961 it was suggested that the Eaton agent might not be a virus and subsequent studies by Chanock succeeded in cultivating an organism for which the term of *Mycoplasma pneumoniae* was proposed.

This study consists of 463 persons who had acute upper respiratory infections on whom the following procedures were done: isolation procedure with culture attempt, complement fixation test and cold agglutination test. Those with outright pneumonia were confirmed by roentgenographic examinations as well.

This relatively large group of patients with proven *M. pneumoniae* has brought about the development of a CF test that is unusually sensitive in specificity.

Most of these patients were in to 10 to 25 year age group; the disease was considered to be rather mild; and all responded quite well to tetracycline therapy.—David C. Alftine, M.D.

METYS, R., ŠNAJDR, V., KRUML, J., and ROUBKOVÁ, H. Pulmonary chondromatous hamartomas. *Med. thorac.*, 1964, 21, 168-186. (From: Medical and X-ray Clinic and Department of Pathology, Institute for Postgraduate Medical Training, and the Tuberculosis Research Institute, Prague, Czechoslovakia.)

Hamartoma was originally defined as a lesion found in the liver although further studies have shown that it more frequently occurs in the lungs. This review consists of 40 observations, 24 chondromatous hamartomas found by necropsy and 16 removed surgically. The authors present detailed studies in a tabular arrangement of all 40 cases and supplement the report by reproductions of pathologic sections as well as roentgenograms.

The microscopic picture shows the predominant structure to be cartilage with the elastic type being most common. Frequently deposits of calcium are found within the cartilage. Changes indicating transition to malignancy were not noted in any case.

The majority of the cases in this review were asymptomatic except in those instances where the lesion was found associated with a disease causing the patient to be examined. Roentgen examination is, therefore, the most important factor in the diagnosis of pulmonary chondromatous hamartomas. Typically they appear as solitary, almost round

shadows of medium density. Their surface is sharp and slightly irregular, ranging in size between 2 and 5 cm. in diameter. In over one-half of them central granular calcifications can be proved. The bronchographic picture is usually without significant change.

Differentiation should be considered from other "coin lesions," including granuloma, tuberculoma, mycetoma, hydatid cyst, solitary metastasis or primary peripheral carcinoma. All of these lesions have the same roentgenographic appearance of a round peripheral shadow, sometimes calcified. According to general statistics, malignant processes represent about 40 to 50 per cent of all coin lesions.

Second to granuloma, chondromatous hamartoma is the most frequent form of benign coin lesion. Therapy requires surgical intervention since this is the only certain way to arrive at the proper diagnosis.

In conclusion, the authors state that chondromatous hamartomas are congenital in their origin and grow to various sizes. They are absolutely benign and clinically asymptomatic. Chondromatous hamartomas are discovered only by roentgen examination, but the diagnosis can seldom be made by the roentgenographic appearance alone.—David C. Alftine, M.D.

HOLMES, BRIAN R. Calcifications of the heart. *J. Canad. A. Radiologists*, Dec., 1964, 15, 163-176. (From: Department of Radiology, Faculty of Medicine, University of Toronto and Toronto General Hospital, Toronto, Ontario, Canada.)

The various types of cardiac calcification tend to have characteristic locations and roentgen appearances.

The advent of surgical techniques for treating many of the diseases in which calcific lesions occur, and the parallel development of better radiologic techniques, make the evaluation significant in assessing such conditions as valvular heart disease, coronary artery disease and some forms of heart failure.

Calcification in the heart is of two types, metastatic and dystrophic.

Metastatic calcification is much less common and is associated with increased availability of calcium ion. It may be seen in such conditions as hyperparathyroidism, hypervitaminosis D and renal insufficiency. It is usually associated with calcification in other organs as, for instance, lungs, stomach and kidney. The deposits are likely to accumulate in tissues with a hyaline structure such as the endocardial elastica of the atrium.

Dystrophic calcification of one form or another is extremely common. It does not require any change in the level of serum calcium and owes its occurrence purely to local pathologic change. It is thought to be related to a low production of carbon dioxide in slowly metabolizing tissue, resulting in a local zone of

relative alkalinity and, therefore, reduced calcium solubility. Thus it can occur in any area of devitalized tissue.

The most efficient way of recognizing the presence of calcium is by means of an image intensifier, and the best method of recording its presence is on cine-film.

Laminagraphy has been described as a method of demonstrating cardiac calcifications. In principle, this also is an inefficient method, inasmuch as the object to be recorded is usually in motion, as are the roentgen-ray tube and the film.

The different structures which calcify generally tend to do so in one of three ways:

- 1.—Plaques (pericardium, atrial wall, thrombi).
- 2.—Linear streaks (coronary arteries, ligamentum arteriosum).
- 3.—Nodules (valves, mitral annulus).

The first group may be seen en face or in profile, the second end-on or at full length and the third in various projections. In identifying the presence, location and nature of a calcification, one must make use of varying patient positions and calcification movements, and must choose appropriate milliamperage and kilovoltage.

Calcification of coronary arteries is probably the most difficult to appreciate.

Detailed techniques for ascertaining different types of calcification in the pericardium, coronary arteries, myocardium, valves, and in aneurysms are comprehensively described and accompanied with numerous illustrations.—*Stephen N. Tager, M.D.*

ABDOMEN

GLANVILLE, J. N. Further experience with the ward acute barium meal. *Clin. Radiol.*, Jan., 1965, 16, 93-95. (Address: St. James Hospital, Leeds, England.)

After going without anything by mouth for 2 hours, the patient drinks 4 to 5 ounces of liquid barium. He is then turned to the right side for 1 to 2 minutes, following which he lies supine on a cassette. A portable roentgenographic unit is used for making an exposure in this position, a mouthful of thick barium being swallowed just before exposing the film. The patient then lies prone with the left side raised 30°, or if examination is made during a blood transfusion, this second film is exposed with the patient supine, and with the right side raised 30°.

In a study of 100 patients checked by laparotomy, gastroscopy or a later barium meal examination in the X-ray Department, there were positive findings in 41 (duodenal ulcer, 15; gastric ulcer, 15; esophageal varices, 2; hiatus hernia, 8; duodenal diverticulum, 1). The findings were negative in 40 patients who probably had acute ulcers. The diagnosis was incorrect in 19 cases, being missed in 13 (gastric ulcer, 3; duodenal ulcer, 9; neoplasm, 1), while false positive findings were reported in 6.

This method is recommended as a method of localizing the site of bleeding in hematemesis where the patient cannot be moved to the X-ray Department.—*Samuel G. Henderson, M.D.*

INDRA, K. J., and CHAWLA, S. Trichobezoar—a case report. *J. Canad. A. Radiologists*, Dec., 1964, 15, 193-194. (From: Department of Radiology, Lady Hardinge Medical College and Hospital, New Delhi-1, India.)

The total number of reported cases of trichobezoar up to 1961 was 240, of which only a few are recorded in the radiologic literature. The authors describe an additional case in detail.

A 10 year old female was admitted to the Lady Hardinge Medical College and Hospital with a history of loss of weight and vomiting, off and on, for 4 months. A mass, approximately 12 cm. by 8 cm., with a smooth surface was palpable in the epigastrium.

A bolus of barium was given under fluoroscopy. The greater and the lesser curvatures were distinctly outlined, showing the mass to be intragastric. Peristalsis was absent. The mass extended into the first and second parts of the duodenum, the third part of which appeared partially obstructed. A diagnosis of trichobezoar extending into the third part of the duodenum was made.

The follow-up roentgenograms showed the barium caught in the mass, giving a mottled and blotched appearance which persisted for 24 hours.

On close questioning, the mother admitted that she had noticed the child occasionally plucking her scalp hairs and swallowing them.

At surgery, a large hair-ball was found, almost completely filling and distending the whole of the stomach, pylorus, first and second parts of the duodenum and forming almost a complete cast of them. The postoperative period was uneventful.

The authors state that most hair-balls are found in females. Symptoms arise only when the ball has reached sufficient size to cause a palpable mass in the abdomen. The usual symptom complex consists of abdominal pain, nausea, vomiting, loss of weight and palpable mass in the abdomen.

The roentgenologic features are often characteristic. A plain roentgenogram of the abdomen may reveal a large mass lying within the stomach and separated from the stomach wall by a thin line of air, the upper part of the mass projecting within the fundal gas. The gastric air bubble is frequently obliterated. Peristalsis is usually absent with marked delay in the emptying of the stomach.

The treatment is necessarily surgical.—*Stephen N. Tager, M.D.*

MORGENSTERN, LEON, FREILICH, MICHAEL, and PANISH, JOEL F. The circumferential small-bowel ulcer: clinical aspects in 17 pa-

tients. *J.A.M.A.*, Feb. 22, 1965, 191, 637-640. (Address: Dr. Morgenstern, 4833 Fountain Avenue, Los Angeles, Calif.)

Recent reports of circumferential small bowel ulceration in adults receiving thiazides and potassium have led the authors to review their experience with 17 of these cases during the past 4 years. Two cases are reported in detail and 15 in summary. Sixteen of the 17 patients were definitely receiving thiazide and potassium drugs and the 17th may also have received them.

The roentgenographic appearance is that of a short stenosing annular lesion occurring in the jejunum or ileum and varying in length from a few millimeters to 4.8 cm. Small bowel obstruction is usual and perforation occurred in 4 patients. The most frequent symptom complex is intermittent cramps, nausea and distention often occurring post prandial. Treatment is segmental resection and in this series produced uniformly good results. Pathologic examination of the ulcer shows nonspecific ulcerative and inflammatory change without evidence of vasculitis.

The authors speculate on the role of thiazide and potassium in causing the ulcer and postulate that the lesion is due to local action of an irritating agent on the mucosal surface with resultant sustained contraction accounting for the circumferential nature of the lesion.—*John A. Tobin, M.D.*

SOUTER, WILLIAM A. Bolus obstruction of gut after use of hydrophilic colloid laxatives. *Brit. M. J.*, Jan. 16, 1965, 1, 166-168. (Address: Formerly, Surgical Registrar, Derbyshire Royal Infirmary, Derbyshire, England.)

The author discusses the obstruction to the large gut from a bolus of hydrophilic colloid laxative, given in all 3 instances described, during a recent postoperative period. Two deaths resulted and the third case recovered spontaneously on conservative treatment.

Apparently the lack of normal peristalsis causes distention of the gut beyond a certain critical level, when the bowel becomes incapable of passing the hydrophilic bolus onward. It would seem that this product should not be used in a recent postoperative period. A survey of the English and French literature has yielded only 2 other similar cases, bringing the total to 5.

The author supports the documentation in each of the 3 cases by roentgenographic illustrations which accompany the article.—*David C. Alftine, M.D.*

DASSEL, PAUL M. Intestinal obstruction—a review of some uncommon causes. *Mich. Med.*, Mar., 1965, 64, 176-181. (From: Department of Radiology, Ferguson-Droste-Ferguson Hospital, Grand Rapids, Mich.)

Paralytic ileus is the most frequent cause of severe small bowel distention, either primary or postoperative. The principal mechanical obstructions are caused by adhesions, small bowel hernias, colonic neoplasms, and diverticulitis. These two large categories probably account for about 85 per cent of all bowel obstructions.

The author reviews a significant group of miscellaneous causes which are uncommon but not rare. Most of these demand prompt surgery for survival of the patient and yet they often result in only moderate or nonspecific symptoms and clinical findings. One must frequently check and constantly keep in mind the causes of intestinal obstruction in order to suspect the possibility of an unusual case when it is encountered.

Seven types of unusual obstructions are illustrated by case presentations. These include: (1) malignant neoplasms of the small bowel, (2) volvulus of the colon, (3) mesenteric infarction, (4) gallbladder perforation, (5) inflammatory strictures, (6) foreign bodies, and (7) fecal obstruction. Surprisingly, fecal obstruction is the most common of these uncommon entities, resulting in 5 per cent of all deaths from intestinal obstruction.—*Arch H. Hall, M.D.*

MARCUS, R., and WATT, J. The radiological appearances of diverticula in the anti-mesenteric intertenia area of the pelvic colon. *Clin. Radiol.*, Jan., 1965, 16, 87-92. (Address: Dr. Marcus, Consultant Surgeon, Clatterbridge Hospital, Bebington, England.)

Recent studies have shown that involvement of the antimesenteric intertenia area in diverticulosis of the pelvic colon is much more common than has hitherto been thought. Several types of lesions can be recognized. These have been described as transverse linear fibrous striae; separation of circular muscle into coarse bundles, showing relative hypertrophy and atrophy of fasciculi; and three types of diverticula—intramural, ridge and saccular. In examination of a series of postmortem specimens of pelvic colons, all of which showed diverticulosis, there was involvement of the antimesenteric intertenia area in nearly 50 per cent of the specimens. Although the earliest visible stage in the development of diverticulosis in the antimesenteric intertenia area is the presence of transverse linear striae, it is only with the development of intramural ridge and saccular diverticula that roentgenologic changes become apparent.

Intramural diverticula invariably occur at the sites of horizontal linear fibrous striae. In barium-filled specimens they produce a spiky outline. The spikes are shorter and the base is wider when the bowel is distended. *Ridge diverticula* present as ridge-like elevations which run transversely in the antimesenteric intertenia area. They represent a further stage in the development of diverticulosis in the antimesenteric intertenia area, and, hence, are fre-

quently associated with horizontal fibrous striae and intramural diverticula. Roentgenologically they also produce a spiky outline. The apex is usually rounded and may balloon to a certain extent. *Saccular diverticula* present as smooth rounded projections, varying in size up to about 1 cm. in height. They are much larger than the usual form of ridge diverticula. As a rule they are located in the lower half of the pelvic colon, and occur in association with intramural and ridge diverticula.

Diverticula in the antimesenteric intertenia area are much smaller than lateral diverticula. The earliest roentgenologic change is a very fine irregularity of outline, corresponding to outward mucosal protrusions at the sites of transverse linear fibrous striae. In the next stage, the mucosal protrusions between the circular muscle bundles, as seen in intramural diverticula, give an appearance of small spikes with pointed apices. Ridge diverticula, occurring as a further extension of the mucosa beyond the muscularis, produce a spiky outline of the same general configuration, but the apices tend to be rounded or ballooned. Saccular diverticula are considerably larger, wide necked, rarely contain fecoliths, and have a smooth rounded outline.—*Samuel G. Henderson, M.D.*

SKELETAL SYSTEM

CLAUS, H. G. Gibt es eine streifige Form der Osteopoikilie? (Does a striated type of osteopoikilosis exist?) *Fortschr. a. d. Geb. d. Röntgenstrahlen u. d. Nuklearmedizin*, Nov., 1964, 101, 522-530. (Address: Institut für Klinische Strahlenkunde der Johannes-Gutenberg-Universität, 6500 Mainz, Germany.)

Osteopoikilosis is a well recognized entity characterized by spotty or sprinkled condensations of bone. It is asymptomatic and without clinical significance. Etiology and pathogenesis are undetermined, but hereditary factors are well established. Constitutional anomalies with associated malformations have also been observed.

A so-called striated variety is rare, having only been described ten times. There is, however, no histologic similarity with the typical osteopoikilosis. Spotty radiolucencies are present between the sclerotic striae, especially in the extremity bones. These are caused by islands of cartilage as in chondrodysplasia. There is also metaphyseal flaring of extremity bones with overgrowth and presence of associated exostoses. The variety is believed to be due to neurotrophic disturbance of bone tissues as in Ollier's disease, having also been called "osteopathia striata" with monomelic involvement. In contrast to the spotty form, hereditary factors have not been verified. Occasionally, associated skin changes, similar to those of the spotty form, are observed, having been called dermatofibrosis lenticularis disseminata.

A so-called mixed form has also been described. It is supposed to be a combination of the spotty and the striated forms. In addition to dense spots, larger longitudinal and ovoid condensations of bone are present. The striae resemble those of melorheostosis. The existence of such a form, however, is very doubtful and inadequately supported as the mixed pattern can be simulated by confluence or by summation of superimposed densities.

It is the author's contention that the striated form is a variety of histologically proved chondrodysplasia, having nothing in common with the spotty osteopoikilosis which is a true condensation of bone. There is no justification for 3 separate forms, and the existence of a striated variety is most improbable.—*Ernest Kraft, M.D.*

CHAWLA, S. Cranio-skeletal dysplasia with acro-osteolysis. *Brit. J. Radiol.*, 1964, 37, 702-705. (From: Department of Radiology, Lady Hardinge Medical College and Hospital, New Delhi 1, India.)

This unusual skeletal anomaly was first described by Hajdu and Kauntze in 1948 under the heading of cranio-skeletal dysplasia. Since then only 3 more cases have been reported in the world medical literature. The author presents a fifth case which illustrates all the features of this rare dysplasia. The patient, a female, aged 33 years, housewife, presented with a history of chronic cough and expectoration for 2 years. The only significant clinical abnormalities were short plump fingers and toes with bulbous ends, and broad curved nails; completely edentulous jaws and hypotonia of all the extremities. Joint hypermobility was particularly evident in the knees and interphalangeal joints. Clinical examination was otherwise normal and full investigation of the blood and urine revealed no abnormality.

Roentgenologic examination demonstrated cystic changes involving the whole of the right lung; these were shown by bronchography to be due to widespread bronchiectasis. The skeletal changes were as follows:

Skull:dolichocephalic, the pituitary fossa being enlarged with a peculiar boat shape and thinning of the clinoid processes; the sutures, especially the lambdoid, were wide with sclerotic margins; wormian bones were present in the occipital region.

Mandible:atrophic with absorption of the alveolar margins; both the maxilla and the mandible were edentulous except for a few unerupted teeth in each.

Lumbar spine:generalized osteoporosis with biconcave vertebrae and abnormal kyphotic curve of the sacrum.

Pelvis: the sacrum was grossly deformed with erosion of the lateral borders; the pelvis was osteoporotic, contracted and triangular in shape; both margins of the symphysis pubis were eroded.

Hands:bulbous swelling of the finger tips with bilateral, asymmetric absorption of the terminal

phalanges, the tips being spared in some fingers; the absorption extended to the center of the middle phalanx of the index finger in the right hand.

Elbows: the right elbow and forearm were normal but on the left side the bones were osteoporotic and hypoplastic, and the radial head was dislocated.

Feet: the terminal phalanges showed similar absorption; the bases of the first distal phalanges were expanded with absorption of their mid-shafts and preservation of their tips; the head of the first right proximal phalanx was atrophic.

Reproductions of a photograph and several roentgenograms illustrating the findings are included.—*Douglas S. Kellogg, M.D.*

LANGENSKIÖLD, A., and RISKA, ERIK B. Tibia vara (osteocondrosis deformans tibiae): a survey of seventy-one cases. *J. Bone & Joint Surg.*, Oct., 1964, 46-A, 1405-1420. (From: Orthopaedic Hospital of the Invalid Foundation, Tenalavägen 10, Helsinki, Finland.)

The authors report 71 cases of tibia vara—a disturbance of growth of the epiphyseal cartilage, epiphysis, and metaphysis at the medial aspect of the proximal tibia, resulting in an angulation into varus deformity below the knee. Two different types have been described; an infantile type and an adolescent type. This report includes 61 cases of the infantile type and 10 cases of the adolescent type.

The infantile type becomes apparent between the ages of 1 and 3½ years. It may be impossible to differentiate it from a marked "physiological" bow leg when initially seen. Microscopic studies show that cartilage necrosis in the zone of resting cartilage is characteristic. Only 2 cases showed spontaneous regression; therefore, the authors feel that surgical correction should not be delayed. The results are best when the surgery is done before the age of 8 years. The various operative procedures used, according to the severity of the condition, are described.

The adolescent type appears at the age of 8-11 years. It differs roentgenographically from the infantile type in that there is no characteristic step-like deformity of the medial epiphyseal line. There is a narrowing of the middle part of the medial half of the epiphyseal plate. After the growth period, the adolescent type can be distinguished from the infantile type only by history. Spontaneous regression may occur in the adolescent type, and surgery is recommended only if the disease shows progression.—*Richard W. Van Buskirk, M.D.*

GOLDENBERG, RAPHAEL R. Chondrosarcoma. *Bull. Hosp. Joint Dis.*, April, 1964, 25, 30-49.

The original classification of the Bone Sarcoma Registry of the American College of Surgeons did not differentiate between chondrosarcoma and the other forms of sarcoma arising in bone. Phemister was one of the first to emphasize the distinction between

chondrosarcoma and osteogenic sarcoma. In 1939 the Committee of the Registry of Bone Sarcoma published a revised classification of bone tumors and accepted Phemister's concept of chondrosarcoma as a distinct malignant bone tumor. It is of interest that MacDonald and Budd found that about one-half of the recorded 5 year cures of osteogenic sarcoma, in the files of the Bone Sarcoma Registry, appeared to represent instances of either chondrosarcoma or fibrosarcoma. A significant contribution was made by Lichtenstein and Jaffe when they defined chondrosarcoma pathologically and established the histologic criteria for the differentiation between benign and malignant cartilage tumors. Their studies established the following histologic criteria when viable noncalcifying areas are examined: (1) many cells with plump nuclei; (2) more than an occasional cell with two such nuclei; and especially (3) giant cartilage cells with large single or multiple nuclei or with clumps of chromatin.

Roentgenographically, the most common site of central chondrosarcoma of bone is the shaft of a long bone, the proximal part of the femur and humerus. Less frequently, the tibia, ilium and a rib have been implicated by the lesion. Approximately half of all peripheral chondrosarcomas involve some part of the pelvis. In the majority of cases of chondrosarcoma of bone, the lesion shows an expanding, somewhat loculated area of rarefaction with scattered, rounded or elongated areas of stippling, calcification or ossification, and the cortices become thinned. The stippling or radiopacities indicate that the tumor tissue matrix is undergoing calcification. Only an occasional case of chondrosarcoma of bone will fail to show the radiopacities which are characteristic of cartilaginous lesions. In those infrequent cases, the lesion is often seen as an expanding area of radiolucency. When the tumor has perforated the confines of the bone, the involved portion of the cortex becomes thickened by new bone. The extraosseous tumor mass will usually show roentgenographic evidence of calcification and even ossification.

In the majority of cases, surgical intervention when done initially should be radical. The policy of preserving one joint ahead of the tumor is a valuable one. A chondrosarcoma in the femur or humerus should be treated by disarticulation of the hip or shoulder joint, respectively. If the lesion has perforated the femoral or humeral cortex and has invaded the soft tissues of the hip or shoulder, then either a hemipelvectomy or a scapulohumeral disarticulation would be indicated. The possibility of recurrence must constantly be borne in mind. The literature does not record any case which was cured by radiation therapy.

The author has observed 22 cases of chondrosarcoma of bone and 5 cases of chondrosarcoma arising in soft tissue. Eight case reports are presented, illustrating the clinical, roentgenographic, gross and microscopic characteristics, and end results of

chondrosarcoma. Several excellent reproductions of photomicrographs and roentgenograms are included.—*Douglas S. Kellogg, M.D.*

NORMAN, WILLIAM H. A child with three lower extremities. *J. Bone & Joint Surg.*, Dec., 1964, 46-A, 1755-1758. (Address: 908 Hume-Mansur Building, Indianapolis 4, Ind.)

This very rare anomaly was present in a female infant who had an extra right lower extremity. There were no other defects. There was a hemipelvis with a leg measuring $9\frac{1}{4}$ inches in length at 11 weeks. The long bones and patella were normal. There was a posterior superior dislocation of the femoral head with a very shallow acetabulum. The foot was deformed and had 3 digits and 3 metatarsals. Two ossification centers were present in the region of the calcaneus.

The limb and hemipelvis were amputated.

There is a discussion of the embryologic formation of limbs. A genetic factor appeared to be excluded and the opinion was advanced that the extra limb was apparently due to a mechanical factor, leading to splitting of the original limb primordium.—*Martha Mottram, M.D.*

HORWITZ, THOMAS. Some pathologic considerations in the diagnosis of lumbar intervertebral disk lesions. *Bull. Hosp. Joint Dis.*, April, 1964, 25, 21-29. (Address: Assistant Professor of Orthopedic Surgery, Indiana University School of Medicine, Indianapolis, Ind.)

The author states that there is no disagreement with the excellence of the results following excision of the intraspinal lesion in the classic case of posterior protrusion or extrusion of intervertebral disk substance. There has not, however, been uniform satisfaction with the sustained results following this procedure in the lower lumbar spine and this has prompted the supplemental use in some clinics of primary spine fusion. An important consideration is that the selection of cases has broadened considerably to include other vertebral lesions which also produce a neurologic deficit and myelographic defect. A consideration of some established findings in intervertebral disk pathology may serve to clarify this further.

First, a posterior disk protrusion is part of a more generalized disk degeneration. Both the anatomist and pathologist recognize a posterior disk protrusion as a part of generalized degenerative lesion that involves the entire intervertebral disk. The pathologist finds degenerative changes in the interior content of a disk space, removed at the time of surgery, even in the acute protrusion with no previous history of backache and a normal roentgenogram. It is noted that a posterior protrusion of a normal intervertebral disk by a single injury must be extremely rare, if it occurs at all.

Secondly, not every intervertebral disk lesion is clinically significant. Degenerative disk disease *per se*, when it evolves slowly as with advancing years, may be associated with little or no clinical manifestation. A localized posterior disk protrusion need not be symptomatic if small and centrally located, especially when there is no nerve root involvement. It is noted that the lumbar neural canal may be encroached upon to a remarkable extent without neurologic complication, the cauda equina being displaceable within a wide range; this is well demonstrated in advanced grades of spondylolisthesis. In accord with this, Lewy in 1949 came to the conclusion from his study that a "bulging disk" should not be excised unless there is definite evidence of its encroachment upon a posterior root.

Thirdly, other lesions, alone or in association with posterior disk protrusion, may contribute to the clinical picture. It is essential to recognize that other lesions, which are largely the result of degenerative processes that involve the intervertebral disk, ligamentum flavum and adjacent bone structures, may produce defects in an intraspinal column of contrast medium that are characteristic of an extradural lesion. The lesions considered are: (a) 'posterior bulging' formed by thin disk substance overlying the hypertrophied or exostotic posterior margins of adjacent vertebrae; (b) projection of the posterior border of the fifth lumbar body behind that of the upper sacrum in association with intervertebral disk degeneration, especially the posterior half; and (c) 'anterior bulging' of the ligamentum flavum due to a marginal bony proliferation of the underlying facets. Jonck concluded from an anatomical study of 400 lumbar spines that when a disk is narrowed it appears that the most likely structures to cause pressure on the nerve root are not the disk itself but the superior articular processes with their overlying ligamentum flavum. Laminar thickening is receiving some attention as an intraspinal space-occupying factor.—*Douglas S. Kellogg, M.D.*

GENERAL

BROWN, JAMES. Primary amyloidosis. *Clin. Radiol.*, Oct., 1964, 15, 358-367. (From: Department of Radiology, Royal Lancaster Infirmary, Lancaster, England.)

In this article the author discusses the roentgenologic features encountered in primary amyloidosis, and reports 6 new cases in which the diagnosis was not suspected clinically or roentgenologically.

Cardiac failure is frequently due to deposition of amyloid within the myocardium. Roentgenologic studies usually show an enlarged cardiac silhouette.

Localized deposits of amyloid tissue within the upper respiratory tract and air passages is the most frequent manifestation of the respiratory form of the disease. The most common form of lower respiratory tract amyloidosis is a diffuse stenosing

infiltration of the trachea and bronchi giving a characteristic bronchoscopic appearance. One may see an indefinite haziness of the lung fields on the roentgenogram, or there may be pleural effusion, or a stippled or nodular appearance, with diffuse accentuation of the bronchovascular pattern.

The roentgenologic findings in gastric amyloidosis may include pyloric narrowing with stenosis or spasm, mucosal atrophy or mucosal hypertrophy, gastric ulceration, gastric stasis and aperistalsis. Golden (1954) described 3 cases of primary amyloidosis of the small intestine, in which he found segments of narrowing and dilatation, coarsening of mucosal folds, mucosal ulceration and very slow transit time of barium.

Amyloid infiltration of bones and joints has rarely been recorded. Bone erosion due to amyloid deposits in the periarticular tissues of the shoulder and elbow joints has been described. Two cases of fracture of the femoral neck due to local soft tissue invasion have been reported. Involvement of vertebrae with wedging and collapse has been described.

Parenchymatous and vascular involvement is common in both liver and spleen and these organs may be considerably enlarged.

Although the diagnosis of primary amyloidosis is usually established by clinico-pathologic investigation, roentgenologic examination may offer assistance in arriving at a diagnosis.—*Samuel G. Henderson, M.D.*

RADIATION THERAPY

VASTERLING, HANS-WERNER, and HILFRICH, HANS-JÜRGEN. Über die prognostische Bedeutung der Temperatursteigerung während der Strahlenbehandlung des Kollumkarzinoms. (Prognostic significance of temperature elevation during radiation therapy of carcinoma of the uterine cervix.) *Strahlentherapie*, June, 1964, 124, 213-218. (From: Universitäts-Frauenklinik, Göttingen, Germany.)

Of the patients treated primarily at the Göttingen University's Gynecologic Clinic for carcinoma of the uterine cervix by radiation alone, the authors studied those who had had temperature elevations to at least 100.4°F. They divided these patients into a group of 216 who had had a fever for no more than 5 days, and a group of 58 who had had a fever for longer than that. Then they matched the patients in each group against "control" patients who had comparable stages of the disease but who had not been febrile during their treatment.

The patients who had had fever of shorter duration had essentially the same course and prognosis as their non-febrile comparison group; i.e., fever of

short duration (and usually without clear-cut cause) did not affect the outcome of the radiation therapy. However, those with fevers of longer duration (the usual cause was inflammation somewhere in the genital tract, often following intracavitary radium) had a 25.9 per cent five-year "cure," while their non-febrile comparison group had a 43.9 per cent five-year "cure;" the respective mortality from carcinoma was 67.2 per cent in the febrile group and 43.9 per cent in the non-febrile group.

Definitive chemotherapy or antibiotic therapy is recommended, but the authors advise against a blanket, prophylactic program of chemotherapy or antibiotic therapy.—*Henry G. Moehring, M.D.*

BONO, F., and DETTORI, P. Aspetti angiografici ed encefalografici nei tumori cerebrali dopo radioterapia con alte energie. (Angiographic and encephalographic findings in cerebral tumors following high energy radiation therapy.) *Radiobiol., radioter. e fisica medica*, May-June, 1964, 19, 192-203. (From: Istituti di Radiologia e del Radio dell'Università di Bologna and Divisione di Diagnostica Neurochirurgica, Ospedale Maggiore di Bologna, Italy.)

The authors have performed carotid and vertebral angiography and encephalography in 20 cases of cerebral tumors of various nature and at different sites. Co⁶⁰ and Cs¹³⁷ teletherapy was used for treatment and the tumor doses varied between 3,500 and 6,000 r. The cases included astrocytoma, glioblastoma multiforme, spongioblastoma, oligodendroglioma, and astroblastoma.

Radiographic studies were obtained before surgery and irradiation and at intervals of 1 month to 1 year; in 1 case up to 3 years.

(a) *Angiography*. The authors conclude that occasionally following irradiation there has been evidence of edema of the hemisphere which was the site of the neoplastic process. There has been no evidence of aneurysmal dilatation, spasm, thrombosis or telangiectasia.

(b) *Encephalography*. In some instances, a moderate ventricular dilatation but no alterations of the cisternal and subarachnoid spaces has been seen.

Recurrences have been demonstrated in areas which were not of the primary tumor and at times in the contralateral hemisphere.—*Anthony A. Blasi, M.D.*

LAMP, J. CURTIS, GRAHAM, JAMES H., URBACH, FREDERICK, and BURGOON, CARROLL F., JR. Keratoacanthoma of the subungual region: a clinicopathological and therapeutic study. *J. Bone & Joint Surg.*, Dec., 1964, 46-A, 1721-1731. (Address: Dr. Lamp, 888 Glenbrook Avenue, Bryn Mawr, Pa.)

Preoperative recognition of keratoacanthoma is important since it responds to local treatment. It is a benign tumor which is found occasionally on the glabrous skin but is altered clinically when it is in the subungual region and confined by the nail plate. The rapid growth within a period of weeks tends to differentiate it from squamous cell carcinoma even though there are some overlapping histochemical features of these two tumors. Patients with keratoacanthoma are younger than those with squamous cell carcinoma, where 80 per cent of the lesions occur after 60 years of age.

Pain, swelling and erythema develop rapidly in the end of a digit. A portion of the nail separates from the bed and then a keratotic crusted nodule develops at the edge of the nail. A white cheesy keratinous material is extruded. There is extensive bone destruction of the phalanx from pressure, with little or no periosteal reaction or new bone formation.

Three cases are reported. The roentgenograms revealed a lytic defect of the distal end of the distal phalanx of the thumb in each instance; in 1 case about 75 per cent of the phalanx was eroded. Clinically they all resembled chronic paronychia, but attempts at drainage were unsuccessful.

The microscopic features of the 3 cases are illustrated; they are identical with those of keratoacanthoma in other anatomic sites and closely resemble squamous cell carcinoma. Virus-like particles have been demonstrated by electron microscopy.

The pertinent differential diagnoses are discussed and include chronic paronychia, epidermoid cyst and osteomyelitis as well as squamous cell carcinoma.

The importance of recognizing these lesions is emphasized because they are benign, and complete removal of the tumor without disturbance of adjacent tissue is indicated. The nail plate should also be removed. Radical surgery or irradiation is inadvisable.—*Martha Mottram, M.D.*

RADIOISOTOPES

McAfee, J. G., Fueger, C. F., Stern, H. S., Wagner, H. N., Jr., and Migita, T. Tc^{99m} pertechnetate for brain scanning. *J. Nuclear Med.*, Nov., 1964, 5, 811-827. (From: Departments of Radiology and Radiological Science, The Johns Hopkins Schools of Medicine, Hygiene and Public Health, Baltimore, Md.)

There continues to be laboratory and clinical investigation of radioactive isotopes in the search for the ideal agent.

Technetium (Tc^{99m}) was first applied clinically for liver scanning in 1963. Thyroid and brain scanning soon followed.

In this report the applicability of Tc^{99m} labeled pertechnetate as an agent for brain scanning is

further investigated. Its tissue distribution in man and animals, and the relative merits of oral versus intravenous modes of administration are discussed.

Data on Tc^{99m} include the physical properties; the source from a molybdenum ⁹⁹-technetium ⁹⁹ generator; preparation of the radioactive material for oral (nonsterile) and intravenous (pyrogen-free) injection; tissue distribution and/or excretion after parenteral and oral administration; body and organ radiation dosimetry; and finally the technique of brain scanning with this element.

From January through June, 1964, 137 brain scans were performed with pertechnetate on 133 patients—52 by oral administration and the remainder by intravenous injection. Adequate follow-up was available on 121 of these patients. There were 22 cases with proven intracranial tumors in this group; 16 were correctly localized, 5 were negative, and 1 was technically unsatisfactory. The over-all diagnostic accuracy was 73 per cent. No marked difference in diagnostic accuracy had been noted in comparative scans between I¹³¹ albumin and Hg²⁰³ chlormerodrin. It appears that certain tumors frequently missed with the other agents, such as low grade astrocytomas and parasellar cysts, may also be missed with pertechnetate.

The short physical half-life of 6 hours, the absence of beta emission, and the gamma emission of 140 kev. permit administration of large amounts of radioactivity (1 to 10 mc); yet the radiation dosage is below the levels obtained with I¹³¹ albumin and Hg²⁰³ or Hg¹⁹⁷ chlormerodrin at standard doses. Procedure and scanning time are greatly reduced while maintaining the same counting techniques used with I¹³¹ albumin or Hg²⁰³ chlormerodrin. With this scanning agent, the vascular anatomic structures of the skull are more clearly seen than with previous agents because of higher radioactive blood stream levels. Extremely high activity is also observed in the salivary and lacrimal glands.—*Richard Pfister, M.D.*

Schmidtke, Ingeborg, Blattner, Liseotte, and Schiessle, Walter. Über einige autoradiographische Beobachtungen nach Inhalation von radioaktivem Yttrium und Zirkonium. (Autoradiographic observations after inhalation of radioactive yttrium and zirconium.) *Strahlentherapie*, June, 1964, 124, 232-241. (From: Medizinische Universitätsklinik, Freiburg/Brsg., Germany.)

Four-month old guinea pigs inhaled nebulized radioactive zirconium oxalate or yttrium chloride solutions for 30 minutes. The animals were killed at varying intervals up to 3 weeks after the inhalation.

Autoradiographic studies (13 illustrations) showed long lasting retention of radioactive zirconium and yttrium in pulmonary interstitial tissue and in regional lymph nodes.—*Henry G. Moehring, M.D.*

CHEMOTHERAPY

HUBACHER, O. Treatment of the terminal stages of cancer with SP-G and SP-I: preliminary communication. *German Med. Monthly*, Sept., 1964, 9, 363-368. (Address: Röntgeninstitut, Scherzligweg 16, Thun, Switzerland.)

This report deals with a group of 107 patients of 442 selected cases of advanced cancer who were treated with the cytostatic agents SP-G (podophylotoxin- β -D benzylidene glucoside) and SP-I (podophyllic acid ethylhydrazide) which act by blocking mitosis in early metaphase. The dose was 400 mg. SP-I or 200 mg. SP-G daily, with total dosages varying widely over a period of months. Some patients had previously been treated with other cytostatic agents while at least one had treatment following surgery and irradiation. The tumor effect in the reported series was judged to be solely due to SP-I or SP-G. Twenty-eight patients (23.6 per cent) were thought to have been successfully treated as measured by objective evidence of either actual shrinkage of tumor, change in blood picture, "significant regression of clinical signs and symptoms," or remissions for at least 6 weeks.

Patients with simple undifferentiated carcinoma of the breast and bone metastases seemed to benefit most, although sporadic cases of other types of cancer, including 1 case of Hodgkin's disease, also seemed to respond.

Diarrhea (30 per cent) and nausea (6 per cent) are the important side effects. The drugs do not cause leukocytopenia.

The author advises that these drugs be used in combination with accepted modes of cancer therapy rather than as a replacement.—*Fredrick S. Mishkin, M.D.*

MISCELLANEOUS

LINDBLOM, ADOLF F. Measuring of doses received by the personnel in radiological departments and its importance. *Strahlentherapie*, May, 1964, 124, 69-74. (From:

Radiological Department of Central Hospital, Växjö, Sweden.)

The author found two situations in which psychologic factors played a big role: (1) The weekly doses received by personnel in diagnostic roentgenology were high because of carelessness, but when film badge monitoring results were conspicuously posted, the amount of carelessness and the weekly doses both dropped considerably; (2) student nurses resist recruitment for training and duty in radiologic departments because of the risk of radiation exposure.

The author's observations show that personnel receive relatively high doses during angiographic and direct, operative cholangiographic procedures—he believes because in each instance protective arrangements are still in their developmental stages.—*Henry G. Moehring, M.D.*

WACHSMANN, FELIX, and KERMANI, DJAWAD. Versuche über die biologischen Wirkungen extrem hoher Dosen. (Experiments on the biologic effect of extremely high radiation doses.) *Strahlentherapie*, May, 1964, 124, 86-98. (From: Institut für Physikalische und Medizinische Strahlenkunde der Universität Erlangen-Nürnberg, Nürnberg, Germany.)

Five series of illustrations depict the effect of roentgen doses up to 12 million roentgens (55 kv., 15 ma., 4 cm. from focal spot, no filter except the 1 mm. beryllium window) on plants' leaves and on the hind legs of frogs.

Up to 500,000 r the effect on a leaf was limited to the irradiated field, but one million roentgens caused the entire leaf, the unirradiated parts included, to wilt. Despite limiting the radiation to non-vital parts (the leg), the frogs died when high doses were given.

These findings, plus a series of experiments in which the irradiated frog's leg was amputated or its circulation greatly slowed by tourniquet, suggest the presence of "radiotoxins."—*Henry G. Moehring, M.D.*

